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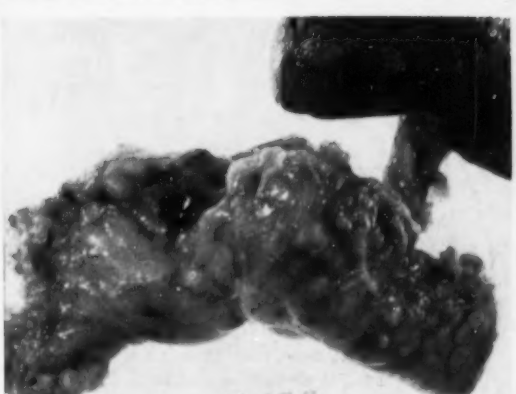

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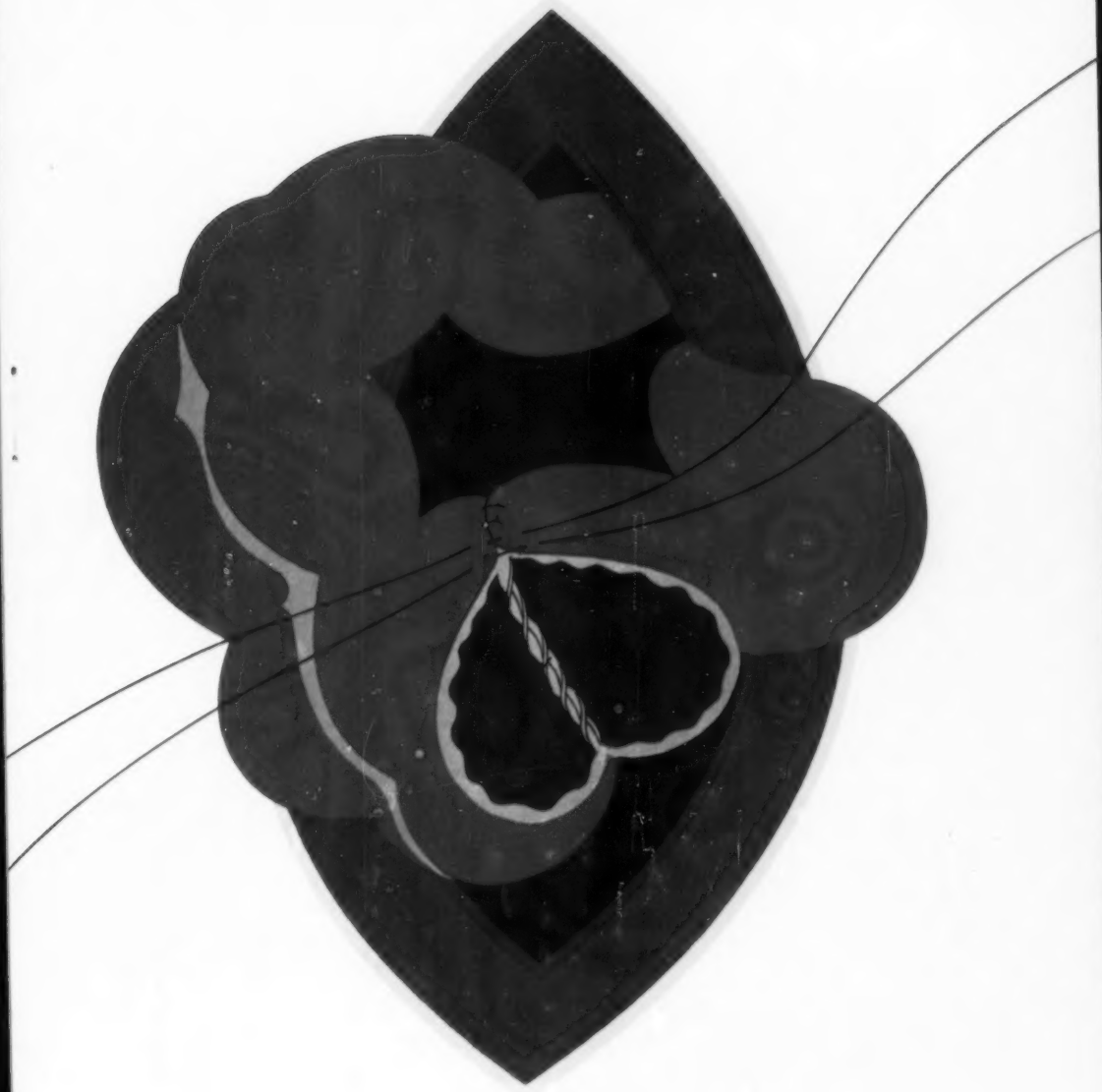

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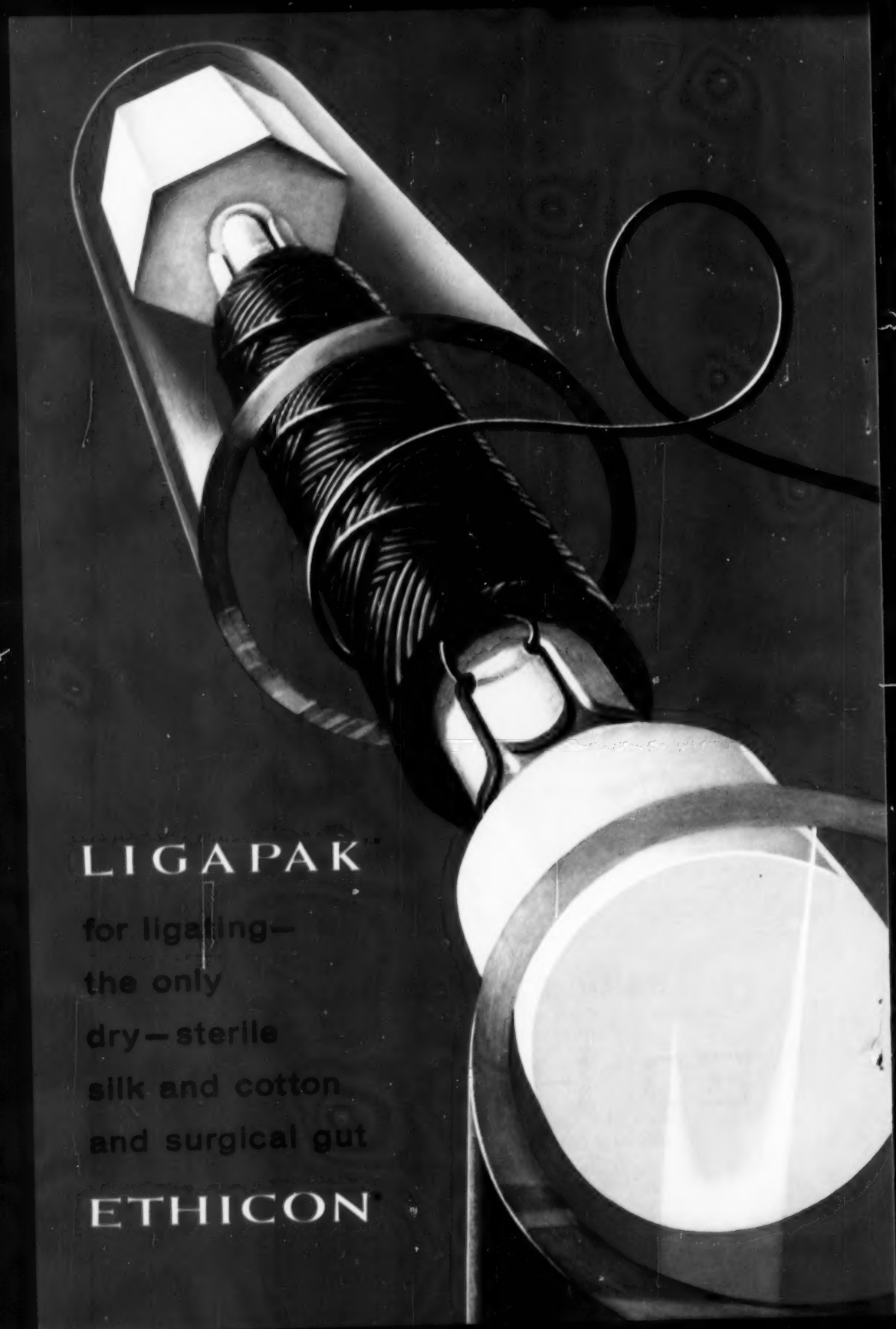
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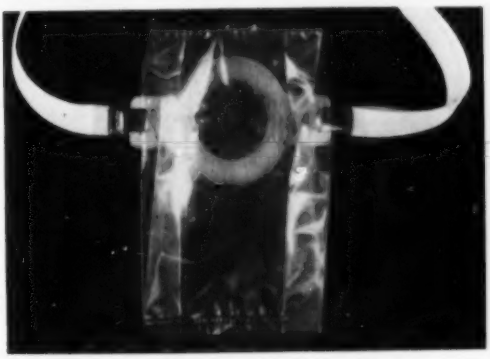

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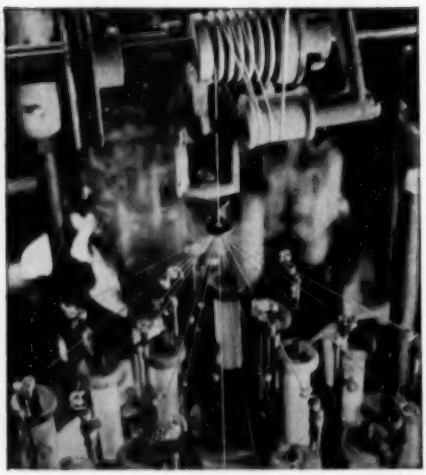

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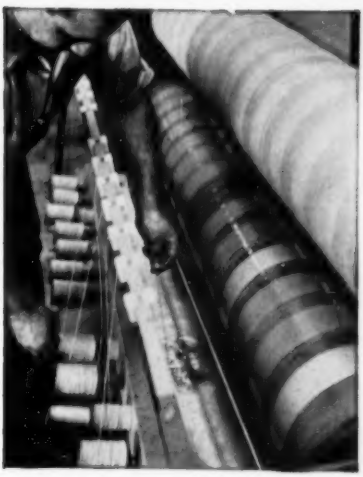

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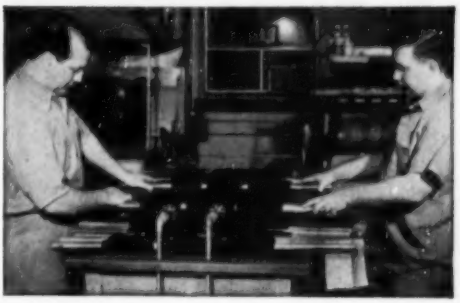

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Control with Corticotropin (ACTH)

CONRAD J. BAUMGARTNER, M.D., Beverly Hills, Calif.
and
JOHN L. REYNOLDS, M.D., Los Angeles

In 1927, Dr. William J. Mayo [1] discovered the first functioning metatastic islet-cell carcinoma, and one year later Roscoe Graham [2] removed the first benign functioning adenoma. Since that time between 400 and 500 case reports of islet-cell tumors have appeared in the literature. By 1950, Howard, Moss, and Quarrie [3] had collected 398 cases, and, by 1952, Whipple [4] reported a personal collection of 34 patients. For the average surgeon, however, this entity will remain a rarity, particularly the malignant tumor, as only about 10% of islet-cell tumors are frankly malignant and about half of these will have functioning metatastases.

The control of the hypoglycemia in such situations not amenable to surgery presents a challenge to the surgeon. We wish herewith to report our experience with one such patient.

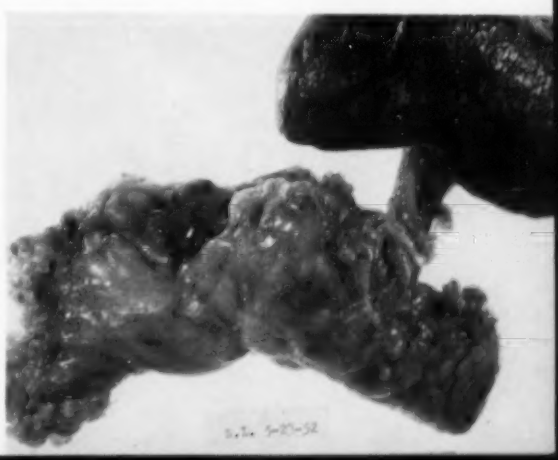
REPORT OF A CASE

Mrs. E. L., aged 59, was first seen by one of us (C. J. B.) in 1944. Oddly enough, her complaint was "dizzy spells." The possibility of hypoglycemia was considered, but it was ruled out by the finding of a fasting blood sugar of 85 mg. She was, however, seen on several occasions in the interim between 1944 and 1952, and dizzy spells were not noted. A small fibroid was noted on these visits. She was again seen May 2, 1952, at which time she was complaining of nausea and severe episodes of hot flashes, associated on one occasion with a dizzy spell. The general examination revealed only a fibroid, which now reached half way up to the umbilicus. X-rays of the stomach, chest, gall bladder, and colon were negative. A hysterectomy was advised at this time, and subsequently, on May 10, 1952, a hysterectomy was performed, with removal of the tubes and ovaries and appendix. During this operation, because of the history of nausea, the upper abdomen was carefully explored and a hard irregular-sized tumor was felt in the region of the tail of the pancreas. The fasting blood sugar determined on the fifth postoperative day was 108 mg.

On May 23, 1952, she underwent reoperation through an upper transverse incision. The liver, gall bladder, and stomach appeared to be normal. There was no ulcer in the duodenum. The tail of the pancreas contained a hard nodular mass to which some omentum had adhered. The pancreas and splenic vessels were severed at the body and the specimen removed with splenic vessels, omentum, and the spleen (Fig. 1).

The pathologist, Dr. Kahler, reported islet-cell carcinoma (Fig. 2) with tumor emboli in the veins within the pancreas (Fig. 3) and metastases to the

Fig. 1.—Carcinoma of islets of Langerhans in the tail of the pancreas. Removed specimen consists of the body and tail of the pancreas with splenic vessels, spleen, and omentum.



From the Department of Surgery, St. Vincent's Hospital, Los Angeles.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 3, 1954.

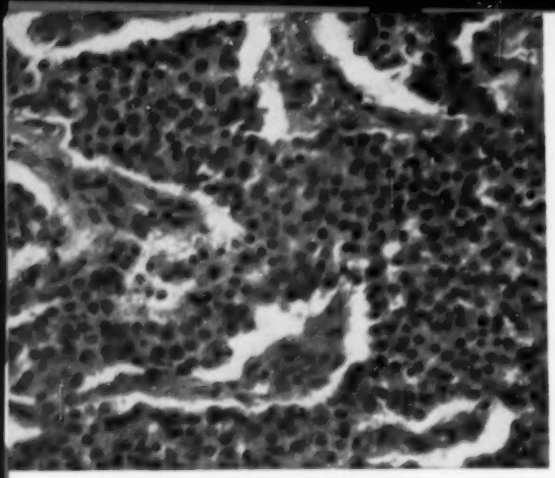


Fig. 2.—Carcinoma of islet cells of pancreas. High power view of tumor cells; reduced to about ⅔ of mag. × 400 in primary lesion of pancreas.

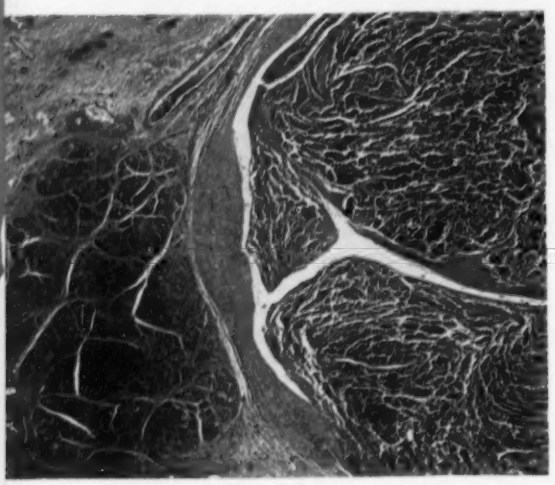
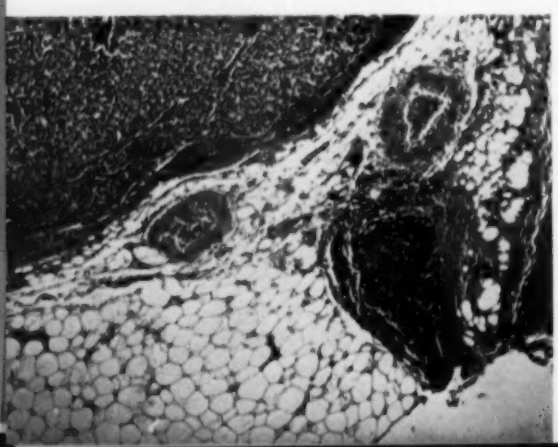


Fig. 3.—Carcinoma of islet cells of pancreas. Tumor cells filling splenic vein within pancreas.

Fig. 4.—Metastases from islet-cell carcinoma to neighboring lymph node; reduced to ⅔ of mag. × 100.



lymph nodes (Fig. 4). An assay for insulin content was not made.

She remained free of symptoms until March, 1953, when she began to experience hot flashes. A fasting blood sugar level was 89 mg. and estrogens were prescribed.

In July, 1953, she began to have episodes of vertigo and some nausea. With these episodes she experienced a numbness of the mouth and tongue. She was instructed to have a blood sugar test made during one of these attacks and also to note if there was any relief attained by the ingestion of food. She did experience relief, and so she deferred the fasting blood sugar test. By September, 1953, the dizzy spells had become increasingly severer and she experienced episodes of diplopia. She was readmitted to St. Vincent's Hospital on Sept. 6, 1953. The pulse rate was 102, blood pressure 140 systolic and 90 diastolic. She appeared very nervous and had a tremor. An irregular liver edge was palpable 4 fingerbreadths below the costal margin. The blood sugar on admission was 33 mg. and the symptoms were promptly relieved by the intravenous administration of glucose. The blood count, urinalysis, and x-rays of chest and skull were negative. Also negative were liver function tests, serum calcium, phosphorus, phosphatase (Bodansky), potassium, chlorides, and CO_2-combining power. A glucose tolerance test showed the results given in Figure 5.

We had become interested in the research work of Stirret[5] in detecting metastatic nodules in the liver by means of radioiodenated human albumin and use of the scintiscanner. The patient was therefore given 2.5 cc. of radioiodenated human albumin intravenously. The resultant scintogram, however, did not reveal any uptake.

A glucose utilization test was obtained by giving 15% dextrose intravenously at a rate of 45 drops per minute. Although there was an initial rise in the blood sugar level (Fig. 6), the level had dropped to normal in six hours and was still dropping when the test was discontinued, a total of 180 gm. of dextrose having been given.

Another operation was performed on Oct. 12, 1953. There was no evidence of local recurrence, but the liver was studded with nodules. At this time the duodenum showed a markedly acute ulcer on the anterior surface. A vagotomy was performed and several nodules removed from the liver. The pathologist reported islet-cell carcinoma with essentially the same cell type as the original (Fig. 7). She was dismissed from the hospital on Oct. 20, 1953, and placed on a high-fat, high-protein diet with in-between feedings and a feeding at 2 a. m. She seemed to be doing very well on this regimen, but on Nov. 23, 1953, she suffered a severe attack of hypoglycemia, with a blood sugar of 42 mg. It became obvious that some other meas-

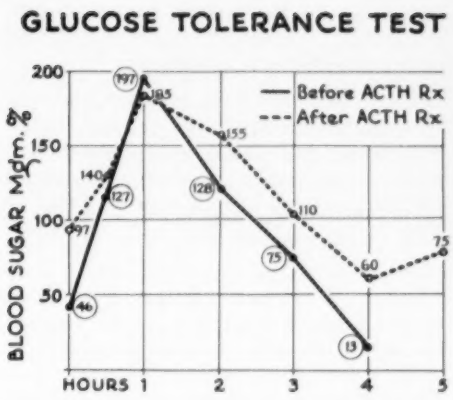


Fig. 5.—Glucose tolerance test showing curves obtained before and after the administration of corticotropin.

ure besides the diet would be necessary to control the hypoglycemia. The drug alloxan was not used because of its variable results and its reported serious side-effects. Because other observers had found that purified corticotropin gel (ACTH gel) was useful in controlling hypoglycemia, it was decided to use it on this patient. She was given 40 units of corticotropin gel intramuscularly daily, potassium chloride by mouth, and a restricted sodium intake. Dramatic relief took place within one week, and the dosage of corticotropin gel was gradually reduced to 40 units every third day. By

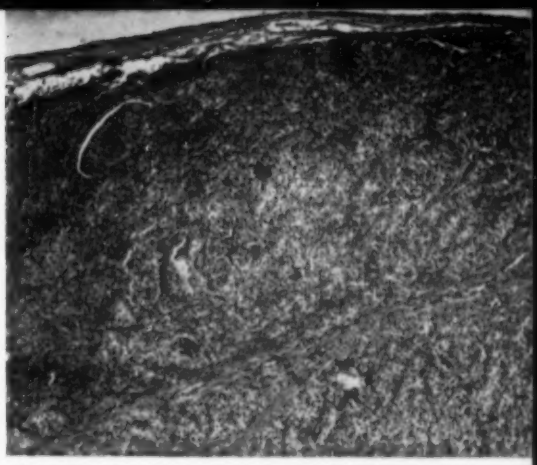


Fig. 7.—Section of biopsy of liver. Nearly all of field is metastatic carcinoma with small area of liver cells; reduced to ⅔ of mag. × 100.

Dec. 16, 1953, she was feeling exceptionally well. The fasting blood sugar was 79 mg., and for some reason we were unable to feel the liver edge.

She was readmitted to the hospital on Feb. 21, 1954, for only one day for further studies. X-rays of the chest were negative and a dextrose tolerance test revealed the findings incorporated on the chart (Fig. 5).

In March, 1954, we decided to increase the interval between injections to 40 units every fourth day instead of every third day. Within one week she had two attacks of hypoglycemia, and the original schedule was therefore resumed. She has continued to remain in excellent health on this routine, working daily as an insurance executive. On Nov. 20, 1954, she returned from an extensive tour of the Eastern States.

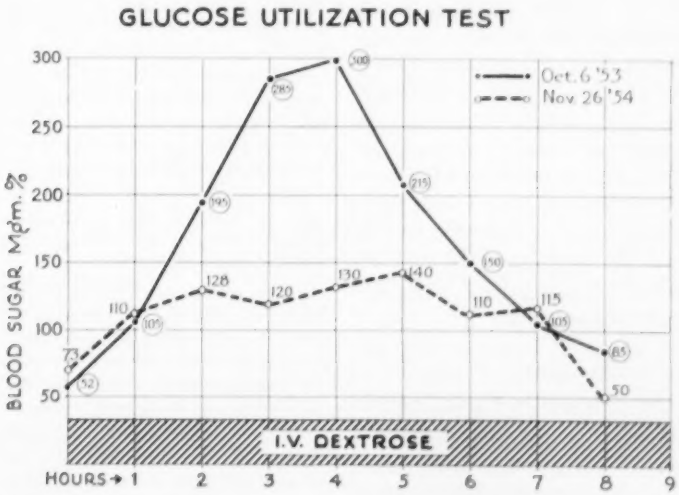


Fig. 6.—Glucose utilization test before and after the administration of corticotropin gel, showing marked utilization of glucose in spite of administration of 180 gm. of glucose (15% dextrose at the rate of 45 drops per minute). Note, however, that there is a marked increase in the test made on Nov. 26, 1954, compared with the same test made on Oct. 6, 1953.

In the interim between November, 1953, and September, 1954, the fasting blood sugar determinations have been as follows:

11/23/53	42 mg.
12/16/53	79 mg.
1/13/54	104 mg.
1/27/54	168 mg.
3/17/54	131 mg.
5/13/54	74 mg.
5/24/54	85 mg.
6/17/54	76 mg.
8/25/54	139 mg.

She was readmitted to St. Vincent's Hospital on Nov. 24, 1954, for studies. The blood sugar on admission was 89 mg. An x-ray of the chest was negative, and the liver edge was still not palpable. A dextrose utilization test was repeated Nov. 26, 1954, and the findings are incorporated on the chart (Fig. 6).

COMMENT

The role of corticotropin is not clearly understood. Thorn [6] stated that corticotropin and cortisone increase glycogen deposition in the liver, increasing glyconeogenesis from amino acids and at the same time opposing the action of insulin peripherally. In a recent (1954) monograph, Kauver and Goldner [7] shared this opinion but stated that corticotropin and cortisone have been used unsuccessfully in islet-cell tumors. Quite to the contrary, however, have been the reports of G. M. Brown [8], McQuarrie [9], Howard [10], H. Brown [11], the British surgeons Smith and Cochran [12], and, just recently, de Peyster and Gilchrist [13], of Chicago. All of these reports dealt with the control of hypoglycemia due to benign islet-cell tumors over a short period of time prior to surgical removal. H. Brown, de Peyster and Gilchrist noted less relief from cortisone than from corticotropin. Mason,[14] a British surgeon, reported control of hypoglycemia of functioning metastases for 25 weeks by the use of 75 mg. of cortisone daily.

SUMMARY

The case herewith reported presents several unique observations: the metastases are functioning while the primary lesion, clinically at least, is apparently nonfunctioning. A duodenal ulcer had developed with the onset of hypoglycemia and was apparently due to hyperchlorhydria produced by hyperinsulinism. To the best of our knowledge, it is the first case in which the hypoglycemia from a functioning metastatic islet-cell tumor has been controlled for a period of one year by corticotropin (ACTH) gel and, lastly, it is hoped that further research with the isotopes may isolate some substance which these functioning nodules will pick up, as does the functioning thyroid nodule with iodine.

DISCUSSION

Dr. Charles W. McLaughlin Jr., Omaha: We are all indebted to Dr. Baumgartner for presenting this very interesting problem, rare as it is. The very dramatic result that he has obtained in controlling the hyperinsulinism from the metastasizing lesion with corticotropin is remarkable.

We have had the dubious privilege of seeing a similar patient during the past two years. This man, 49 year of age, was admitted with the classic symptoms of hyperinsulinism, lightheadedness, dizziness, and fainting spells. He was found to have low blood sugar reading of approximately 30 mg. per 100 cc. and after complete study came to exploration. In the tail of the pancreas a 6-cm. islet-cell adenoma was found.

No metastases were present, but the pathologist reported this to be an islet-cell adenoma with a bizarre cell type and made a diagnosis of probably a malignant tumor. Pathologists reserve that right from their experience with this type of lesion.

The man's blood sugar returned to normal in the immediate postoperative period and remained so for approximately two months. He then again began to experience episodes characterized by lightheadedness and dizziness. Subsequently he would become almost maniacal and, weighing 260 lb. and being very strong, it took six of our better interns to confine him during the period of time in which we would give him intravenous dextrose.

Reexploration was done, and at the second operation we found innumerable nodules distributed throughout the liver. The primary site in the pancreas felt negative.

In the postoperative period the low blood sugar readings continued. We were at a loss to know how to manage this man, since obviously he would be an institutional problem. We considered (as did Dr. Baumgartner) alloxan and decided against it on the basis of experience elsewhere.

While we were debating this matter, the attacks became less frequent. The man insisted that he be dismissed from the hospital. Having five good-size sons, who agreed that they would take care of him with the help of a practical nurse next door, he was dismissed. To our amazement, the attacks have become less frequent. They now occur about

every three days, always preceded by an aura, allowing time to take carbohydrate.

He is controlled entirely by taking large quantities of reinforced fruit juice. His weight has reached the level of 280 lb., but he continues to work at his job as a salesman for furnaces. He does not drive a car, but his wife serves as his chauffeur. I have no explanation for this rather dramatic remission without therapy. We shall certainly try corticotropin if he does not continue to do well.

DR. LOGAN LEVEN, St. Paul: I wish to report a case of a woman, aged 57, first seen in the spring of 1944. She gave a history of periods of amnesia and unconsciousness for a period of about one year. Blood sugar levels had been noted down to 47 mg. On May 12, 1944, a resection of the tail of the pancreas and a splenectomy were carried out for and islet-cell adenoma of the tail of the pancreas, measuring 3 in. in diameter. At the time of operation gallstones were noted in the gall bladder.

The patient was relieved of her symptoms. Because of epigastric distress, a cholecystectomy and appendectomy were performed on Nov. 18, 1947. In September, 1948, it was noted that she had some dizzy spells. However, fasting blood sugar was 104 mg. per 100 cc. In November, 1950, it was noted that she had spells of weakness unless on a high carbohydrate diet. Her fasting blood sugar was 70 mg. per 100 cc. Hypoglycemic attacks continued and became more frequent, and on March 8, 1951, the fasting blood sugar was 38 mg. per 100 cc. On March 15, 1951, a 6-mm. adenoma was removed from the body of the pancreas. This proved to be an islet-cell adenoma. In the immediate postoperative period the blood sugar levels were normal but within a week had dropped to 65 mg. She continued to have hypoglycemic attacks and on Aug. 2, 1951, a resection of the tail and body of the pancreas was carried out. No tumor was noted. There were numerous adhesions in the region of the liver. When the liver was exposed, multiple nodules could be seen. Two of these were removed and the pathologic report showed metastatic islet-cell carcinoma of the liver. Blood sugar levels postoperatively fell from 102 mg. down to 68 mg. per 100 cc. one week after the operation.

At this time alloxan therapy was considered. Bailey had shown experimentally that permanent diabetes could be caused in the experimental animal by administration of alloxan. Because alloxan was a toxic substance that may produce liver necrosis and a fatal outcome, it was decided not to use it in this case. Others have shown that while alloxan may destroy normal islet cells, the islet-cell adenomas appear resistant to its action.

On Nov. 15, 1951, this patient was started on oral cortisone acetate, 25 mg. three times a day. This did not adequately control her symptoms. On Dec. 10, 1951, she was admitted in hypoglycemic shock with a blood sugar of 27 mg. per 100 cc. On Jan. 28, 1952, cortisone (Cortone) acetate, 100 mg. three times a day subcutaneously, was started. On this therapy blood sugar levels above normal were adequately maintained. The amount of cortisone acetate was gradually reduced so that at the end of one week, she was carried on cortisone acetate orally. By July, 1952, the patient had gained 30 lb. of weight and had considerable swelling of the face, hands, and feet. On July 13, 1952, the patient was again admitted to the hospital. The blood sugar level was 38 mg. per 100 cc., and it was decided to start corticotropin therapy. Fifteen units three times a day was given. She was sent home on 40 units of corticotropin (Acthar) gel daily. She developed rather marked glycosuria and was put on diabetic diet. The blood sugar level in August was 193 mg., at which time she was being carried on 8 units a day of corticotropin (Acthar) gel. She continued this same dosage for a period of about one year, when she again developed hypoglycemic attacks. She was admitted to the hospital and placed on 60 units of corticotropin daily for a few days and sent home on a regimen of 24 units of corticotropin daily. In January, 1954, she developed pain in the right shoulder and between the shoulder blades. X-rays of the spine at that time were negative. The liver was palpably enlarged. Liver function studies were normal. Dextrose tolerance tests showed a diabetic curve. Within a month she developed numbness and weakness of her legs. Spinogram on Feb. 8, 1954, showed a complete block at T1 and T2 disc level due to an extradural mass. On that day a laminectomy was carried out, with removal of a tumor mass in the ventral aspect of the dura. The pathologic report was metastatic islet-cell carcinoma. This was followed by x-ray therapy, and the patient has got along very well since then, recovering the use of her extremities. At the present time she is being carried on 12 units of corticotropin gel daily and shows an occasional trace of glycosuria. Her blood pressure is 180/90, with her weight 186 lb.

MR. TOBY LEVITT, c/o British Consulate-General, Chicago: Gentlemen, the first case we listened to was a most memorable one, and I feel very fortunate at being present to hear it. The second two cases were also very important.

Here we had an incidence of a pancreatic islet-cell tumor with a definite embolic spread to the vessels, which we saw, a definite spread via the lymphatic to the lymph nodes, and we know that those were lymph nodes because they showed the triad of signs. You could see the capsule of the lymph nodes, you could see the nodule sinus, and you could see the lymphoid follicles.

In addition, we had multiple spread by metastases to the liver, so that according to the present concept the dice were definitely loaded against the patient.

We note that a scintogram of the liver was made. Here I rather plead ignorance. I should like to know from Dr. Baumgartner the validity of the test he described, as to the function of these metastases in the liver.

Secondly, I should like to know from him, when he removed the ovaries, why he removed them, and what he found, because to me it is rather important.

Further, we have this most important fact, namely, the dramatic relief by corticotropin or cortisone in this instance, as an accessory to primary surgery.

I should like to know from Dr. Baumgartner, or any other member here, how and why this relief was obtained. Is it due to suppressive effect on the pituitary, which then does not produce its stimulating effect on the pancreas, or is it due to some other reason?

I know Dr. Baumgartner and the two following speakers reporting their cases will follow up these histories in the same effective manner in which they have done their preliminary studies, because it is only through such clear and good records that we will eventually be able to understand these very difficult problems.

DR. CONRAD J. BAUMGARTNER, Beverly Hills, Calif.: Dr. Davis, I should like to show a 50-ft. reel of film on another case which we encountered about a year ago. While the projectionist is preparing the film, I wish to make a few comments. Several of the authors mentioned discovered that cortisone did not work as well as corticotropin gel in controlling the hypoglycemia. We felt that corticotropin gel should be given, as it absorbs more slowly and a single dose would last over a longer period of time.

Mr. Levitt asks why the scintogram. The work of Dr. Stirrett was reported before this Society within the past year. His work was carried out at the Wadsworth Veterans Administration Hospital in Los Angeles. Dr. Stirrett injected radioiodenated albumin intravenously and demonstrated that there was an uptake of the radioiodenated albumin by metastatic nodules in the liver in some cases. These metastatic nodules were demonstrated by the scintogram and their presence later proved by the surgical department. We thought we might be able to demonstrate metastases in our particular patient by the use of the scintillator, but such was not the case.

Surely there must be some substance which these highly specialized islet cells will pick up. That is why I make the plea that someone in the larger institutions take an interest in this type of research to determine if a substance could be found that these cells would pick up, as the thyroid takes up irradiated iodine.

Mr. Levitt's second question was, "What proof do we have that the metastatic nodules were functioning?" I repeat, as I stated in my paper, at the time of the last exploration we found no evidence of recurrence in the region of the pancreas, but the liver was absolutely studded with nodules and we therefore assumed that the nodules in the liver were the ones that were functioning.

The third question, "Why did I remove the ovaries?" One might say they were just in the way [laughter], but she was a woman past the menopause, and in such a situation the ovaries are frequently removed with the uterus, particularly so when the uterus is as enlarged as this one was. Also remember that she was 59 years of age. To the best of my knowledge, the ovaries were absolutely normal and I believe the pathologist reported them as being so.

I wish now to show the film. It should be of sufficient interest and you may enjoy seeing it.

[The film was shown while comments were being made.]

This woman, we felt, had irreversible brain damage from repeated states of hyperinsulinism from an islet-cell tumor. She was stuporous and had to be fed by the nasal tube for a month. The blood sugar was low (18 mg.) and she was constantly going through the gyrations which you see. She was accepted in our department with considerable apprehension. We were informed by the medical department that she no doubt had irreversible brain damage and that complete deterioration would occur if surgical intervention was deferred.

In the operating room the question arose as to what type of anesthesia should be used. The anesthetist refused to give a spinal, as there had just been a law suit over a spinal anesthetic in which the plaintiff received a large sum of money in settlement from the defendent anesthetist. Intubation gas anesthesia was therefore given. During the time of surgery, the patient was continuously so unrelaxed that it was impossible to work or be relaxed; there was dark blood. You will notice in the picture that the blood becomes very black every once in a while. A point of criticism may arise at this time: why did we not remove the distal half of the pancreas with the tumor? Simply because she was so obese (260 lb.) and unrelaxed and we could not see, but could feel, the tail of the pancreas as far up as the spleen. We did, however, suture the pancreatic bed very well, although it was almost necessary to transect the pancreas to get the tumor.

You now see the tumor, and the last scene shows the removed benign islet-cell tumor being cut.

During closure of the abdomen, this patient had a temporary cardiac arrest, but pulsation was resumed with massage through the diaphragm. She left the operating table in a relatively good state, with a normal blood pressure. She was taken to the ward and placed in bed. The nurse was unable to get the pulse, and the resident was called. On his arrival, a few minutes later, she was pronounced dead.

I believe her death may be attributed to the inhalation anesthesia. This woman had irreversible brain damage from repeated states of hyperinsulinism. The cerebral centers were already knocked out and the administration of something which primarily affects these centers simply resulted in the apnea and dark blood which we encountered. I am of the strong opinion that the patients with cerebral damage should not be given an anesthetic which affects the brain but should be given spinal anesthesia, which has no effect on the already damaged brain.

I feel our anesthesia department was influenced by the recent law suit in which someone had sued for $100,000, and had collected. I recall the anesthetist's making a statement to the effect that we were going to lose the patient on the table and he was not going to be the one responsible for her death. I definitely feel that improper selection of the anesthetic resulted in repeated states of anoxemia, cardiac arrest, and death in this patient after she was returned to the hospital ward.

DR. WALTMAN WALTERS, Rochester, Minn.: I apologize for arising again this morning, but I do so because of a principle that I think is exceedingly important in surgical or medical meetings. If the essayist finishes his discussion with an addendum and there is no opportunity to discuss it, an erroneous impression may be left with the listeners.

Dr. Baumgartner, I believe that if you will use ether for general anesthesia a few times for operations for hyperfunctioning adenomas of the island of Langerhans you will come to prefer general anesthesia. Ether in these cases mobilizes the glycogen from the liver, and therefore you do not have to be so careful about sustaining a normal physiologic level of glucose in the blood by intravenous injection. I believe that is important.

I do not know the cause of death in the last case reported by Dr. Baumgartner nor do I know how the death could have been prevented, but certainly the appearance of the blood, as Dr. Baumgartner described it suggests that anoxia probably played an important role.

In speaking now I do not wish to appear to criticize Dr. Baumgartner but merely to bring out that a case of this sort should be discussed whenever it is presented even though discussion of the paper is completed.

I should like to say too that recently I operated upon a man aged 46 years and removed a single hyperfunctioning recurring metastatic islet-cell carcinoma of Grade 1 and 2.5 cm. in diameter from the right lobe of the liver, which was palpable at operation. This patient had had three previous operations elsewhere on the pancreas, one for removal of the primary islet-cell tumor, the second, a partial pancreatectomy, and the last one a radical Whipple (April 11, 1951) which removed the head of the pancreas (all of the pancreas that remained) and the duodenum. Corticotropin in a dose of 20 mg. was given intramuscularly on March 27, 1951 (two weeks prior to the Whipple operation), without benefit to the hypoglycemic reactions. After the Whipple operation the patient received 20 mg. of corticotropin in 1,500 cc. of 10% solution of sodium chloride as an intravenous drip in a 24-hour period on May 31, 1951.

On June 30, 1953, he received 300 mg. of cortisone intramuscularly. The following day and each day thereafter for a total of seven days he was given 200 gm. of cortisone intramuscularly. Beginning on July 8, he was given 400 mg. of cortisone orally each day for five days. The patient was given alloxan intravenously from November, 1953, to January, 1954. None of these seemingly influenced the hypoglycemia or the symptoms resulting from it for any period.

If corticotropin controlled the hypoglycemic reactions of Dr. Baumgartner's patient with multiple metastatic islet-cell tumors in the liver and did not in the Mayo Clinic patient I referred to, the difference in results may be due to the fact that the clinic patient did not have a pancreas and Dr. Baumgartner's patient did. Moreover, when I took out the single metastatic islet-cell carcinoma, a reaction developed like that which occurs after removal of a primary islet-cell tumor of the pancreas, namely, temporary hyperglycemia. The value for blood sugar was 432 mg. per 100 cc. the day after the operation. This gradually fell and varied from 65 mg. to 35 mg. during the next two weeks. With the low values for blood sugar the patient again began to have mild symptoms, such as disturbance of vision and sweating. Thinking that I might have overlooked a second metastatic tumor in the liver, I reexplored his liver two weeks later, on Aug. 6, 1954, without finding any additional tumors, but there was a subdiaphragmatic abscess, which I drained. His blood sugar reading was 45 mg. per 100 cc. on dismissal, Oct. 5, 1954.

On Dec. 1, 1954, a letter from his wife reported the patient as having concentrations of sugar in the blood varying from 94 to 106 mg. and that he was taking 12.5 mg. of cortisone daily and feeling fine. The patient wrote on Jan. 5, 1955, to give the exact dosage of corticotropin and cortisone that he had received before coming to the clinic and he also stated that he was doing well.

I apologize again for speaking twice this morning but I think it is important when a controversial question arises for somebody to get up and call attention to it, not with the idea of criticising but merely so that an erroneous impression will not occur with regard to a method or an interpretation.

DR. CONRAD J. BAUMGARTNER, Beverly Hills, Calif.: I thought I was all through. [Laughter]

Dr. Walters, I did not intentionally throw that film in at the end so that nobody could talk about it. I was afraid of the hook that pulls one out at the end of 10 minutes, and I knew I would not be able to show it during the allotted time given for my paper; so I placed it in with the discussion.

Regarding the spinal anesthetic, I still definitely feel that the woman would have been better off with a spinal rather than with an inhalation anesthesia, because she already had anoxemia of the brain from hyperinsulinism. This resulted in permanent damage to the brain. We know that gas anesthesia affects the cerebral centers, whereas spinal anesthesia does not. The anesthetist did have her intubated and he was trying to give her sufficient oxygen, but the minute he would begin to give more gas the blood would turn black. He was a resident, and perhaps someone with more experience could have done better. I wish that we had had another anesthetist, but under the circumstances I believe the outcome would have been the same. I do not know how it came about, but I am convinced that a spinal would possibly have saved the day.

It probably is fortunate that the patient died. She would have been a "vegetable" and a great burden to her family and the taxpayer from then on.

In the original discussion I failed to answer all of Mr. Levitt's inquiries. He asked for an explanation of the effect of corticotropin in controlling hypoglycemia. According to Thorn, it acts in a manner like robbing Peter to pay Paul, that is, by converting amino acids into glycogen. The internists call it glyconeogenesis. Thorn and others also state that corticotropin inhibits the effect of the insulin peripherally. Just how this takes place I do not know. The co-author wrote his thesis on corticotropin and the corticosteroids. He, no doubt, would be able to answer your questions. I begged him to come along to discuss this phase of the paper, and I recall making the statement that "somebody is going to ask that question and the senior author will not be able to answer it." His reply was, "you can handle it yourself."

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The Noble Plication Procedure

Application to Acute and Chronic Recurrent Small Bowel Obstruction

GORDON K. SMITH, M.D., Los Angeles

The most frequent cause of small bowel obstruction is chronic adhesive peritonitis following abdominal surgery. The patient who has had recurrent attacks of acute obstruction presents a difficult and distressing problem to the surgeon. Such a patient at operation has extensive adhesions with matted loops of small bowel. After enterolysis, extensive damage to visceral and parietal peritoneum results. Avoidance of further episodes of obstruction in this patient lies not in the attempt to prevent adhesions but in controlling adhesions certain to form.

In 1937 Noble [1] applied the principal of plicating the bowel, forming loops in such a fashion that kinks and obstruction might not develop. Subsequent observers, notably Seabrook,* Poth,[4] and Robert Smith [5] have reported their experiences with this procedure and were well satisfied with the results.

During the year 1949, the surgical services of the University of Southern California School of Medicine established policies for the management of acute small bowel obstruction. Primarily these policies consisted of early surgical intervention, operative decompression of the severely distended bowel, and attempts to peritonealize raw surfaces developing during enterolysis. Those patients who have had repeated attacks of small bowel obstruction due to chronic adhesive peritonitis with extensive damage to visceral or parietal peritoneum have been considered candidates for plication procedures.

PERSONAL EXPERIENCES WITH PLICATION

I should like to report the application of these principles to 39 patients with small bowel obstruction who were seen in private practice since the year 1949. Thirty-six patients with acute small bowel obstruction are included in this report. Twenty-three of the patients with acute obstruction had strangulating mechanisms and eight required resection because of nonviable segments. Of these 23 patients, 10 had a history of two or more previous laparotomies for obstruction. Eleven of the 36 patients with acute obstruction had total plication; 13 partial plication, and 8 resection. Three additional patients with chronic small bowel obstruction who underwent total plication are included.

PRINCIPLES OF MANAGEMENT

Preoperative decompression by means of intubation was begun as soon as the diagnosis of obstruction was suspected. The Miller-Abbott and Harris tubes have been almost abandoned during the past two years. I believe that preoperative decompression is more rapid and complete with a large Levin tube than it is with the Miller-Abbott or Harris tube. Precious time can be lost in attempts to pass the long tubes into the small bowel. The size of the lumen is too small for adequate drainage and, with the suction tip below the pylorus, gastric drainage is also frequently necessary by means of a Levin tube.

Decompression of the severely distended small bowel at the time of surgery was done

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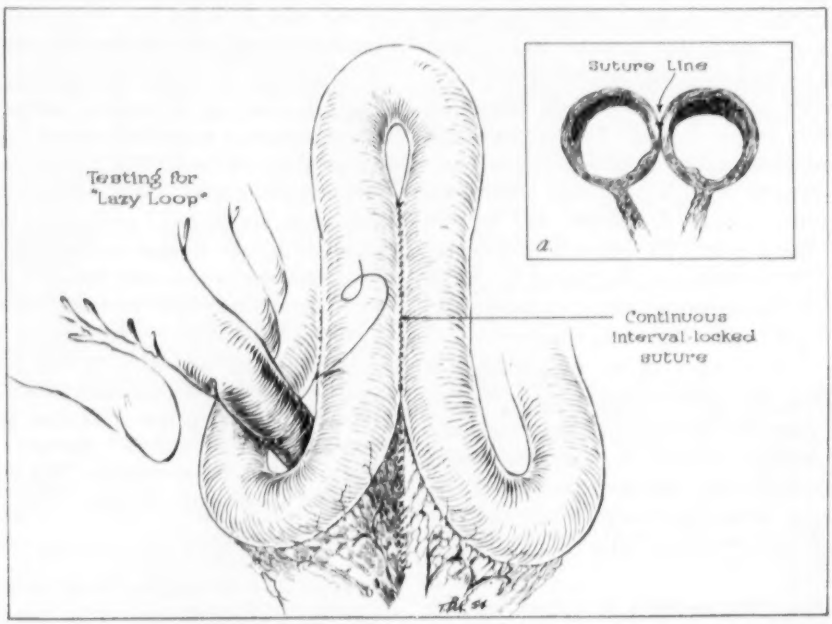
From the Department of Surgery, University of Southern California School of Medicine and the Hospital of the Good Samaritan.

* References 2 and 3.

in 16 patients. When resection was done, the small bowel was decompressed at the point of resection by the use of the ordinary Mayo suction apparatus. During the past year, the Cantor [5] long suction has been available, and this has been used for decompression of the small bowel either through a stab wound at a point in the small intestine or through a stab wound in the cecum, the suction apparatus being passed cephalad through the ileocecal valve. I believe this latter method to be the procedure of choice whenever technically possible. I am firmly convinced that suction decompression contributes to a lower morbidity and mortality in small bowel obstruction.

Plication was performed with the use of a continuous interval-locked chromic (gut) suture placed midway between the mesenteric and antimesenteric borders. It has been shown that sutures placed at the antimesenteric border or at the mesenteric attachment may lead to obstruction by a loop of bowel becoming trapped between distended neighboring loops of plicated bowel. Interrupted sutures may pull out, thus leading to "fiddle-string" adhesions between neighboring loops as reported by Seabrook or to perforation of the bowel as reported by Robert Smith. In the partial plication group the leaves of the mesentery were closed to prevent herniation of small bowel through these potential foramina.

Plication procedure was used in all patients where extensive damage to visceral peritoneum was present. The decision as to whether partial or complete plication should be done depended upon the amount of damage to the peritoneum. Where the damage was isolated to a short segment of bowel, partial plication was deemed adequate. Where "skip" areas of normal bowel intervened or extensive loss of visceral peritoneum was present, complete plication was done.



Principles of technique of Noble plication. Continuous interval-locked chromic suture approximating bowel and mesentery, leaving "lazy loop." Insert shows approximate site of suture in bowel wall.

REPORT OF TOTAL NOBLE PLICATIONS DONE FOR ACUTE SMALL BOWEL OBSTRUCTION

CASE 1.—L. K., 36-year-old woman. Six previous laparotomies, three for obstruction. Admitted April

10, 1954, with strangulating obstruction due to volvulus of terminal ileum. Extensive loss of parietal and visceral peritoneum. Suction enterostomy for enormous distention. Peritoneal fluid and exudate marked. Total Noble plication.

Case 2.—T. O., 56-year-old woman. Three previous laparotomies, two for obstruction. Admitted Dec. 21, 1953, with strangulating obstruction of terminal ileum. Fifty centimeters of ileum resected; extensive visceral peritoneal loss. Suction decompression at point of resection. Total Noble plication. Developed pelvic abscess which subsided under antibiotic therapy.

Case 3.—J. L., 36-year-old man. History of previous appendectomy. Admitted Jan. 2, 1953, with strangulating obstruction over band adhesion. Extensive visceral peritoneal loss. Total Noble plication.

Case 4.—M. W., 47-year-old woman. History of two previous laparotomies, one for obstruction. Admitted March 4, 1953, with simple obstruction of ileum. Large amount of visceral and parietal loss. Total Noble plication.

Case 5.—M. R., 38-year-old woman. History of two previous laparotomies, one for obstruction. Admitted March 4, 1953, with simple obstruction of ileum. Large amount of visceral and parietal loss. Total Noble plication.

Case 6.—N. P., 40-year-old man. History of two previous laparotomies, one for obstruction. Admitted Dec. 31, 1952, with acute simple obstruction of terminal ileum. Extensive loss of visceral and parietal peritoneum. Total Noble plication.

Case 7.—R. K., 11-year-old boy. Laparotomy on May 6, 1953, for traumatic rupture of left kidney. Soiling of peritoneum with urine. Complete simple obstruction of ileum 11 days postoperatively. Extensive loss of visceral and parietal peritoneum. Total Noble plication May 17, 1953.

Case 8.—M. D., 50-year-old woman. History of four laparotomies; two for excision of retroperitoneal sarcoma. One Feb. 13, 1953, strangulating obstruction midileum. Extensive parietal and visceral peritoneal loss. Resection of ileum. Total Noble plication.

Case 9.—B. A., 43-year-old woman. History of two previous laparotomies, one for obstruction. Admitted June 22, 1954, with strangulating obstruction of terminal ileum. Large amount of peritoneal fluid present; extensive visceral and parietal peritoneal loss. Suction enterostomy. Total Noble plication.

REPORT OF PARTIAL NOBLE PLICATIONS REQUIRING SECONDARY PROCEDURES

Case 10.—M. R., 29-year-old woman. Total hysterectomy, right salpingo-oophorectomy July 6, 1954. Complete peritonealization except for raw area on sigmoid where free omental graft was placed. Complete small bowel obstruction developed July 13, 1953. Miller-Abbott tube passed into jejunum did not relieve obstruction. Laparotomy on July 13, 1953, revealed volvulus where ileum was adherent to site of free omental graft on sigmoid. Suction cecostomy of small bowel; complete Noble plication because of extensive peritoneal loss on ileum and pelvic peritoneum. Patient has remained well.

Case 11.—R. K., 41-year-old woman with history of total hysterectomy. Admitted in 1948 with strangulating obstruction of eight hours' duration. Laparotomy Nov. 6, 1949, revealed terminal ileum adherent to pelvic floor with volvulus at this point. Partial Noble plication of three loops and peritonealization of pelvic floor. Patient remained well until July 30, 1951, when she developed complete simple obstruction of terminal ileum. Extensive loss of visceral and parietal peritoneum required total Noble plication. Patient has remained well since.

Case 12.—G. E., 34-year-old woman with history of two previous laparotomies. Operated on July 6, 1953, for strangulating small bowel obstruction of 72 hours' duration. Enormous distention of bowel present. Suction enterostomy at point of obstruction. Partial Noble plication performed. Patient developed oliguria, became confused and removed her Levin tube. Extensive fluid loss and distention. Reoperation on July 12, 1953, for secondary mechanical obstruction. Upper jejunum was adherent to pelvic peritoneum with complete obstruction. Nonviable bowel where it had undergone volvulus. Resection of 30 in. of jejunum and end-to-end anastomosis. Suction decompression at point of resection. Patient died 12 hours postoperatively. Postmortem findings were fibrinous peritonitis, lower nephron nephrosis, and chronic rheumatic mitral heart disease with mitral stenosis.

REPORT OF TOTAL NOBLE PLICATION REQUIRING SECONDARY PROCEDURES

Case 13.—M. B., 41-year-old woman. Strangulating obstruction of upper ileum on Jan. 5, 1954. Suction enterostomy done. Partial plication of two loops performed. Because of recurrent cramping pain and distention, laparotomy was done on April 19, 1954, which showed dense adhesions between the terminal ileum and parietal peritoneum at site of previous suction enterostomy. Lysis of adhesions, leaving extensive raw visceral and parietal peritoneal surfaces. Total Noble plication performed. Developed hypoproteinemia. Peristalsis did not return until the 12th postoperative day following intravenous administration of serum albumin. Patient complained of persistent symptoms similar to those prior to the second laparotomy. Explora-

tion on July 14, 1954, showed several loops of terminal ileum to be separated except for "fiddle-string" adhesions. End of second distal loop was adherent to sigmoid, producing angulation and partial obstruction. Adhesions lysed; bowel replicated with chromic absorbable (gut) sutures. Recent small bowel study failed to reveal obstruction. Symptoms of distention and occasional cramping are now completely relieved by bethanechol (Urecholine).

CASE 14.—R. K., 24-year-old woman. Acute obstruction of ileum on Feb. 25, 1951, with partial plication of raw surfaces of ileum and right oophorectomy for corpus luteum cyst. Symptoms of intermittent obstruction began in 1952 and became severe in 1954. Several x-ray studies showed no evidence of obstruction. Laparotomy done on April 8, 1954. Three long fibrous adhesive bands were found running from the terminal ileum to the pelvic floor. These were lysed and, because of raw areas on pelvic floor and ileum, total plication was performed. Patient made uneventful convalescence and had no symptoms until Oct. 7, 1954, when she was readmitted with a strangulating obstruction of upper jejunum. Laparotomy revealed a loop of jejunum about 24 in. below the ligament of Treitz had prolapsed between two "fiddle-string" adhesions and undergone volvulus of 360 degrees. Bowel was gangrenous; resection and end-to-end anastomosis were performed. Most of jejunum was separated except for "fiddle-string" adhesions. Ileum appeared to be adherent from previous Noble plication. Patient made uneventful recovery.

COMMENT

The 12 patients with acute small bowel obstruction who had total Noble plications are well and asymptomatic. Two of the three patients operated on for chronic obstruction had additional surgery. In both of these cases, the loops of bowel were widely separated and joined only in places by so-called "fiddle-string" adhesions. One of these had a revision of the plication because of separation of the loops, producing partial obstruction. The second had a strangulating obstruction of the upper jejunum six months after total plication.

The only additional complication of any magnitude was the occurrence of a pelvic abscess, which developed in one patient who had small bowel resection and total plication.

Of the 13 patients who had partial plication 3 required further surgery. The first of these was a patient who, in addition to partial plication, had a free omental graft placed over a raw area on the sigmoid. Secondary strangulating obstruction was found at the point where the free omental graft had become absorbed. The second patient was asymptomatic for two years after partial plication and returned with simple obstruction which required laparotomy. Following enterolysis, peritoneal damage was so severe that total plication was performed. Surgery in the third patient was delayed for some 72 hours after the onset of her symptoms. This delay is believed to have been a factor contributing to her death.

The degree of postoperative distention has been variable and at times troublesome. In some cases return of peristalsis was delayed for 8 to 10 days. X-ray evidence of increased small bowel gas persisted up to six weeks after plication.

Perhaps the fact that the operation seems to have been more successful in the presence of acute obstruction suggests there was the added factor of low-grade peritonitis, which aided in the agglutination of the loops of small bowel. In the two cases of total plication that had additional surgery, one showed enormous postoperative distention which may have contributed to separation of the loops of bowel. However, the second patient who returned with a strangulating obstruction had the least amount of postoperative distention of any in this series. Studies are under consideration to determine the optimum type of suture material and the feasibility of utilizing a foreign material in addition to the suture, in the hope of establishing a firmer adhesive process between plicated loops of bowel.

Of the 11 patients who had total plication when acutely obstructed, 6 had strangulating obstructions and all had extensive damage to visceral peritoneum.

SUMMARY

The Noble plication procedure was found to be a valuable adjunct to the treatment of acute bowel obstruction in patients with severe damage to visceral peritoneum.

In addition to contributing to a low morbidity and mortality, it offers the patient far greater protection against recurrent small bowel obstruction than enterolysis alone.

The selection of patients for elective surgery with intermittent or recurrent small bowel obstruction presents a distinct problem. The results of the plication procedure in this series were better in patients with acute small bowel obstruction than in those with chronic obstruction.

I believe the best results are to be obtained in patients with such extensive peritoneal damage that recurrent obstruction seems likely to develop.

Plication of the small bowel is not without hazard. The possible morbidity attendant upon this operation, in my opinion, contraindicates its use as a prophylactic procedure except in carefully selected cases.

It is hoped that the results of experimental procedures and further clinical reports will increase our knowledge regarding the technique and the indications for the plication procedure.

1136 West Sixth St.

DISCUSSION

Dr. Caleb Smith Stone Jr., Seattle: My interest in the plication operation is limited to its application in the treatment of patients who have acute small bowel obstruction or who suffer from long-standing chronic obstruction.

At operation on such a patient one encounters adhesions so extensive that the necessary enterolysis leaves widespread areas of mesentery and bowel denuded of serosa.

In this group of chronic sufferers, recurrence of obstruction may be anticipated unless some definitive procedure can be accomplished.

Since 1946 at the Mason Clinic we have had experience with 15 such patients treated by this operation. In this group of 15, only 1 patient was a man; 14 were women. Multiplicity of former operations was the rule, and most of them had had previous pelvic surgery.

Two-thirds of the group had been previously operated upon one or more times because of acute obstruction; in fact, operations for acute obstruction constituted almost half of the total number of 54 operations performed on this group of 15 patients prior to plication.

Eleven of the 15 patients were contacted one year ago. The remaining four, however, were well when last heard from.

In the group followed up one year ago, five have enjoyed complete relief of symptoms. They enjoy a normal diet, regular bowel habits, and have no complaints. Five patients complain of occasional mild cramping associated with distention, but by their own statement consider the result satisfactory. One patient who was a narcotics addict at the time of plication remains addicted to narcotics and reports no benefit at all from his operation.

Pertinent postoperative difficulties have been limited to mild cramping and moderate distention in a few patients. There have been no deaths in the group. Two additional patients have been operated upon satisfactorily during the past year, too recently, however, to be included in a follow-up study.

The technique we have employed is essentially that described by Dr. Smith. We believe that the plication operation offers the most satisfactory means now available by which we may relieve the symptoms of the chronic sufferer and prevent the recurrence of acute obstruction in this highly selected group of unfortunate people who have widespread chronic adhesive peritonitis.

Dr. Robert Sydney Smith, Boise, Idaho: My experience with the Noble plication operation for small bowel obstruction has not been as extensive as that of Dr. Gordon Smith. Since 1950, I have carried out this procedure in 13 selected cases, and only 3 of these were acutely obstructed at the time of surgery. Ten patients were suffering from chronic recurrent intestinal obstruction, and each of these had had multiple operations previously.

I would like to present one case which illustrates some of the pitfalls and limitations of the Noble plication principle. In this case, I had my first experience with intestinal plication, the operation being performed more or less as an act of desperation in a 54-year-old woman who was unrelieved after 12 operative procedures for small bowel obstruction.

At surgery on June 14, 1950, in a four-hour-long procedure, I freed up all small bowel adhesions, dismantled a malfunctioning ileocolostomy, resected 3 ft. of ileum, and plicated the small intestine from the ligament of Treitz to the ileocecal valve.

Two points in my technique proved subsequently to have been in error. I approximated the loops of bowel with interrupted fine nonabsorbable (cotton) sutures, and I extended the operation to [illegible] loops of upper jejunum which had been [illegible] free of adhesions. After four months of freedom from any abdominal complaints whatsoever, this patient, after taking a laxative, developed an acute obstruction under an omental adhesion. This omen-

tal band was adherent to the descending colon at the site of the previous ileocolostomy that had been taken down.

In a relatively short time the plicated loops of jejunum were literally blown apart by intestinal distention. Jejunal leakage and a fatal peritonitis resulted.

Now I would like to show two slides to illustrate the mechanisms which caused death in the case presented.

[Slide] This first photograph shows the gross appearance of the jejunum at autopsy. The intestine shows constriction and localized ecchymosis at the point at which it had been obstructed by an omental band. A hemorrhagic area indicates a second site of avulsion of a nonabsorbable (cotton) suture and perforation of the bowel to permit a leakage of bowel contents. In profile, a nonabsorbable (cotton) suture is seen still attached to the bowel, with one avulsed end hanging free.

[Slide] This photomicrograph shows a section from the wall of the obstructed jejunum. A nonabsorbable (cotton) suture is seen in cross section with a marked cellular reaction of inflammatory type in the surrounding tissues. It is difficult to say at this time whether this inflammatory process was present during the patient's entire four month postoperative course or whether this was secondary to changes at the time of the acute obstruction.

After having changed my plication technique to provide for the employment of running 000 chromic absorbable (gut) sutures and the avoidance of plication of the upper intestine when this bowel has not been denuded of its peritoneal covering, I have had results which are, on the whole, very satisfactory. I have performed, in all, four more total small bowel plications and eight partial plications in the past four years.

None of these patients has required secondary abdominal surgery to the present time.

Dr. Raleigh White, Temple, Texas: I would like to express my appreciation to the membership for the opportunity of attending this meeting and for the privilege of discussing this splendid paper by Dr. Smith on the Noble procedure.

The Noble plication procedure utilizes the principle of external fixation of the bowel by suture to prevent kinks that might lead to further obstruction. An alternate procedure that I wish to mention to you is one that I used first personally in 1948, at which time I was casting around for something to relieve these patients of the possibility of subsequent obstruction.

At that time, after freeing the bowel surgically of all adhesions, I splinted the entire small bowel by a long intestinal tube threaded from the stomach down through the terminal ileum to the ileocecal valve, in the hope that by holding the bowel in large loops and without angulation, the adhesions that form will be strong enough after 10 to 12 days to permit removal of the tube, thus maintaining the large coils in a fixed position and thereby preventing further episodes of intestinal obstruction.

The happy result in that first case prompted me to use it subsequently, and I have used it ever since. I mention this merely to state that now three to five years have elapsed since the first 12 cases were treated and a recent follow-up shows that none of these patients has had to have subsequent surgery.

A follow-up by mail indicates that at the present time they are all doing reasonably well. I get the impression, however, that there have been some gaseous distention at intervals in some of these patients, but it has not been a problem necessitating hospitalization.

[Slide] This is the artist's conception of splinting the entire small bowel, after release of adhesions, on this long intestinal tube. The tube is passed preoperatively past the ligament of Treitz down to the first point of obstruction. I use a gadget that you may recognize. It is the Meyer vein stripper, to help pass the tube on down into the small bowel by one or two small enterotomy openings during the operative procedure.

[Slide] This shows the procedure being done at the operating table.

[Slide] This is the artist's conception of what we hope to accomplish in these cases, namely, to allow the denuded bowel to form large and more or less symmetrical loops on a semirigid long intestinal tube, allowing fixed adhesions to form, and hoping that acute kinks will not recur postoperatively.

I think the method may have some additional merit for two reasons. One is that it can certainly be done quickly. It does not take long to do this procedure. That may be of interest and value in these poor-risk patients.

The second reason is control of postoperative distention, which Dr. Smith mentioned as a problem. You must admit that it makes for a good night's sleep to have the feeling that this entire denuded small bowel will be splinted on a long intestinal tube and will prevent as far as possible early obstruction in the postoperative period.

I know of nothing more distressing to the surgeon and to the patient, to say nothing of an anxious family, than to have obstruction remain after surgery for the correction of such obstruction.

Dr. Allen Marston Boyden, Portland, Ore.: Dr. Smith asked me to discuss his paper because I had taken part in a symposium on this subject at the Pacific Coast Surgical Association meeting a year ago.

My interest was originally stimulated by Dr. Seabrook, of Portland, Ore., who reported this

procedure in 1948. During the five years prior to January, 1954, my associates and I at the Portland Clinic had performed this operation a total of 23 times in 21 patients.

We elected this procedure for one primary reason, namely, marked denudation of the small bowel at operation. The adhesive process was found incidentally in many of our patients who were operated on for other abdominal disease. All but one of our patients had had at least one previous operation, the majority having had more than one.

Seven plications were performed for acute obstruction; 15 were performed incidental to other major abdominal operations, such as second- and third-stage colectomy, resection for ileitis, and hysterectomy after pelvic surgery. Only one was done for chronic obstruction, mentioned as a common indication by Dr. Smith and Dr. Stone.

We had one death in this series which could well have been related to this surgery. This was a young man who, after a total colectomy (except for the rectum) for ulcerative colitis, developed intestinal obstruction on the 16th day. At operation there were widespread adhesions and the loops of bowel were markedly distended. I am sure that some of his disease had progressed into the ileum. He died, on the 20th day after plication, from multiple perforations of his bowel, many of which were not at points of the suture but simply at points where the adhesions had been divided.

All but four of our patients have been followed for a period of six months to four years. Only one has had a recurrence of obstruction. We have had none who have had any pertinent symptomatology otherwise.

The patient who had recurrence of obstruction had ulcerative colitis. After total plication, the loops of ileum had become separated, and one of them was attached to the abdominal scar. Plication was again performed. The patient remained well despite still another operation necessitated by extension of ulcerative disease in the remaining colon.

Our technique has differed from that described, since we have used interrupted sutures. Aside from the above experiences there has been no difficulty.

In conclusion, I am convinced that this procedure has great merit and is often life-saving in patients in whom large raw areas of denuded small bowel remain after operation for either adhesive obstruction or other intra-abdominal disease. It will prevent subsequent obstruction in approximately 95% of these patients.

Small bowel fistula is a complication which can occur and which did occur in one of our patients and in five patients in one of the other series reported. Certainly, plication should be reserved for those patients with very extensive serosal damage. To apply it prophylactically for minor degrees of adhesive disease is unwise. The operation is necessarily time-consuming and shocking and may contribute to a mortality already high because of the severity and extent of the primary disease.

We should thank Dr. Smith for reappraising an operation which as yet has not gained the acceptance that it deserves.

DR. GORDON K. SMITH, Los Angeles: I should like to thank Drs. Stone, Smith, White, and Boyden for their comments. I think Dr. White's method is a most ingenious one. However, there are some things that I wonder about. Dr. Seabrook told me some time ago that in two patients he had had tremendous difficulty in removing Miller-Abbott tubes that had been placed down prior to the plication. Apparently Dr. White has not had that trouble.

In one patient that I had early, when I was interested in this problem, I cut the tube off at the nose and let it go down that way and was able to recover it. Actually, the time involved in the actual plication procedure is nothing compared to the time involved in the complete enterolysis of the bowel; so far as the time element is concerned I would not let that bother me.

Concerning Dr. White's comment about being absolutely sure he would have no distention, it has been my experience many times with patients with long intestinal tubes, passed far below the pylorus, that I have had to additionally pass a Levin tube through the other nostril to decompress the stomach. I think Dr. Payne, formerly of Minnesota, has written a recent article on this and has devised a tube which passes just through pylorus into the duodenum, having a suction tip both in the duodenum and in the stomach. It is his feeling that the air which produces distention is swallowed air.

I neglected to say that the mortality in my series, fortunately, was limited to the one patient who died after the partial plication.

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Congenital Obstruction of the Small Intestine and Colon

B. G. LANNIN, M.D.
N. LOGAN LEVEN, M.D.
and
LYLE A. TONGEN, M.D., St. Paul

Congenital obstruction of the small intestine and colon occurs much more frequently than has been generally realized. Considering atresia alone, the incidence, according to Webb and Wangensteen,[16] in 1931, was about 1 in 20,000 births. However, some 20 years later Evans,[4] after an extensive review of the world's literature, estimated the incidence to be about 1 in 1500 births and further calculated that this condition resulted in the death of about 3000 babies annually in the United States and of more than 50,000 in the rest of the world. Wilson, Nelson, and Harshbarger[16] have estimated the number of children that are lost each year in the United States is about 2200. They further state that this latter figure is approximately the same as the number of deaths (through 55 years of age) from appendicitis in the United States in 1949 and also equals the deaths from rheumatic fever in all ages. The significance of the above statement is obvious and emphasizes the need for improvement in the management and mortality of these infants.

There are additional causes of obstruction besides atresia. The others which will be considered in this report are volvulus of the midgut with malrotation of the colon, meconium ileus, and one extremely interesting case of congenital intussusception.

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COMPLETE ATRESIA OR INCOMPLETE ATRESIA (STENOSIS) OF THE INTESTINE AND COLON

Because of its common etiologic origin, this group of cases will be considered together. However, the various locations of the lesions will be found in Table 1. The embryological failure of vacuolation and resulting reestablishment of intestinal lumen determine the site and whether the persistent defect will be complete or partial. This theory has been accepted by most authors with the possible exception of Evans.[4]

The commonest site in this small series was the duodenum. However, Gross,[7] who undoubtedly has had the largest experience in this country with these infants, observed a slightly higher incidence in the ileum. The jejunum, colon, and multiple areas of involvement are less common.

TABLE 1.—*Location and Distribution of Lesions in Small Bowel and Colon*

Type and Location	No.	Living	Dead
Atresia of duodenum	7	5	2
Stenosis of duodenum	5	3	2
Atresia of jejunum	2	1	1
Absence of ileum and cecum	1	0	1
Stenosis of colon	1	1	0
	16	10 (62%)	6

Clinical findings in these infants are quite typical and have been well described. Vomiting almost invariably occurs within the first day of life, and progressive distention will develop, depending upon the site of the obstructing lesion. With low obstructions, the vomitus will be fecal in odor and tremendous distention may develop.

A flat plate of the abdomen taken within the first day or two is ordinarily sufficient to establish the diagnosis. The differential

diagnosis is of somewhat academic importance, as, with the possible exception of meconium ileus, once the diagnosis of obstruction has been made, exploration is advisable and the site is relatively unimportant. Farber's [5] test of the meconium may prove to be a useful but not essential aid in determining the completeness of the obstruction. We agree with most authors * that barium by mouth is not only unnecessary but may be actually harmful because of future difficulties with aspiration, inspissation, and evacuation.

TREATMENT

The importance of the preoperative care of these children cannot be overemphasized. The actual operative procedure is only a small but obviously highly important step in the successful management of these infants. Preoperative and postoperative gastric suction, careful and judicious administration of parenteral fluids, transfusions, vitamins, and antibiotics are all essential and do not need further emphasis. The improvement in anesthesia in the past few years has undoubtedly added materially in the reduction of mortality and morbidity in these infants, and we have been extremely fortunate in the past few years in having the services of skilled anesthesiologists.

We customarily employ a long muscle-splitting rectus incision and believe that careful but complete evisceration of the intestinal tract is necessary for accurate diagnosis. The technical maneuvers necessary to perform an anastomosis in these infants are well known to members of this audience. Our preference is for a one row, open side-to-side anastomosis, using nonabsorbable suture for the duodenal lesions. In the jejunal and lower small bowel obstructions an end-to-end anastomosis is customarily employed. We have also observed that the lower obstruction cases have a longer convalescence and period of adjustment and may exhibit partial chronic distention for many months.

* References 3 and 14.

A brief case history of each patient follows.

R. K., a white boy, born April 29, 1945, with a birth weight of about 7 lb. This child started vomiting within the first 24 hours of life but passed several small meconium stools. On May 2, a small swallow of barium was given by mouth, which showed complete atresia in the first portion of the duodenum. There had been no bile noted in the vomitus, but there was bile in the meconium. On May 3, exploratory laparotomy was carried out, at which time a complete atresia was noted at the junction of the first and second portions of the duodenum. In addition the colon was not completely rotated, and the mesentery was rather loosely attached. A duodenojejunostomy was carried out, and the child made an uneventful convalescence and was dismissed from the hospital on June 8, 1945. The child was last seen on July 18, 1947, at which time a normally functioning anastomosis was observed between the duodenum and small bowel, with normal emptying of the stomach. The child was developing normally in all respects.

L. T., a white girl, born Jan. 22, 1946, with a birth weight of 6 lb. 4 oz. This child also showed persistent vomiting of all her feedings within the first 48 hours, and x-ray examination was done on Jan. 25; at this time a small amount of barium was given, revealing complete obstruction of the second portion of the duodenum. An exploratory procedure was done on the same day, at which time it was noted there was a complete atresia of the duodenum with the jejunum being extremely small, with a diameter of about 4 mm. A duodenojejunostomy was carried out, after which the child made a very satisfactory recovery and was dismissed from the hospital on Feb. 24, 1946. She was last seen on Dec. 15, 1947, at which time a gastrointestinal x-ray was made which showed an enteroanastomosis in the duodenum which functioned satisfactorily.

J. D. S., born Jan. 28, 1948, at another hospital. The child was premature and the birth weight was 3 lb. 9 oz. The child started vomiting within the first few hours, although it was observed that the child had several small meconium stools. The child was carried on intravenous and subcutaneous fluids but failed to gain weight and was transferred to the Childrens Hospital on Feb. 3, 1948, at which time the weight was 2 lb. 14 oz. The diagnosis of prematurity and high intestinal obstruction was made, and the child was prepared with transfusions, plasma, and intravenous feedings. On Feb. 4 exploration was carried out, at which time a complete atresia of the first portion of the duodenum was observed. The small bowel was found to contain bile, and a duodenojejunostomy was carried out. The child did fairly satisfactorily for the first three postoperative days but died rather suddenly

on the fourth postoperative day. Postmortem examination was done, and the essential findings were congenital atresia, complete, of the duodenum; duodenojejunostomy; hemorrhage at the anastomosis; hemoperitoneum; hypertrophy and dilatation of the esophagus, stomach, and duodenum, and prematurity.

J. S., a white girl, born Feb. 2, 1948, with a birth weight of 6 lb. 4 oz. This child was born after a normal delivery but shortly after birth, when fed, vomited bile-stained fluid. X-ray examination was done, showing a huge dilated stomach and duodenum, and a diagnosis of congenital atresia of the duodenum was made. The child was transferred to the Childrens Hospital, and on Feb. 6, 1948, exploratory laparotomy was carried out. A complete atresia of the third portion of the duodenum was observed, with bile being aspirated from the stomach. A retrocolic duodenojejunostomy was carried out, after which the child made a satisfactory convalescence and was dismissed from the hospital on March 14, 1948. She was last seen on April 15, 1948, at which time she had had no vomiting and was gaining satisfactorily.

L. G., a white girl, born Sept. 6, 1949. The child's birth weight was 5 lb. 15 oz., and it had appeared normal for the first two days of life. However, the child then started to vomit and subsequently several coffee-ground emeses were observed. The child was admitted to the hospital on Sept. 8, 1949. Physical examination revealed a well developed, well nourished, slightly jaundiced newborn girl. X-ray examination of the upper gastrointestinal tract revealed complete obstruction of the duodenum just distal to the duodenal bulb. On Sept. 9, she was taken to surgery, at which time a complete atresia of the duodenum was noted above the ampulla. The stomach and first portion of the duodenum were found to be markedly distended. An anastomosis was then done between the first portion of the jejunum and the stomach. The child seemed to do fairly well after surgery but died on the first postoperative day. The essential autopsy findings were complete atresia of the duodenum, with hypertrophy of the proximal portion, recent anastomosis which was intact, and acute aspiration pneumonia.

L. A., white girl born March 12, 1952. Vomiting of material some of which was bile-stained was observed within the first 48 hours. On March 16, 1952, a flat plate of the abdomen showed the first and second parts of the duodenum and the stomach markedly dilated and filled with gas. A small mixture of iodized oil (Lipiodol) in liquid petrolatum was injected, revealing a complete stricture at the junction of the second and third parts of the duodenum. Exploration was carried out on March 16, at which time an atresia was noted in the duodenum in the region of the superior mesenteric vessels. A retrocolic duodenojejunostomy was carried out, after which the child made a satisfactory convalescence and was discharged from the hospital in good condition. However, she was again admitted to the hospital, on May 9, 1952, with a history of recurrent vomiting for the past 48 hours; she had no stools in the past 24 hours. A flat plate of the abdomen was made, which showed many loops of distended small bowel with no evidence of air in the colon. Nasal suction was instituted; repeat examination on May 10 and May 11 showed no improvement in the degree of distention in the abdomen, and on May 11 reexploration was done. At this time it was observed that approximately 2 ft. of the distal ileum had become adherent and rotated 180 degrees, forming a strangulation obstruction of this portion of the bowel. The strangulated segment was excised and a one row open anastomosis made in the distal ileum. The child made an uneventful convalescence after this procedure and was dismissed from the hospital on May 18, 1952, weighing 9 lb. 3 oz. She was last seen on Aug. 25, 1952, at which time she was developing satisfactorily. The parents moved to another city.

J. M., a white girl, born Jan. 25, 1954, with a birth weight of 4 lb. 8½ oz. During the first day of life this child developed typical symptoms of upper intestinal obstruction, and a diagnosis of atresia of the duodenum was made. Unfortunately, in addition this child exhibited typical findings of mongolism. On Jan. 27 duodenojejunostomy was carried out, after which the child made a good convalescence but was not able to be discharged from the hospital until April 28, 1954, because of an ulceration on the buttocks which developed from an intramuscular injection. At the time of discharge the child weighed 7 lb. 3 oz. and has not been seen since.

Baby D., a white boy, born Feb. 14, 1949. This child had been born by Caesarean section and weighed 6 lb. 8½ oz. at birth, the indication for section being incipient toxemia of pregnancy. After delivery it was observed that he vomited all feedings and showed progressive abdominal distention. He was admitted to the Childrens Hospital on Feb. 17, 1949. Exploratory laparotomy was done on Feb. 17, at which time a stenosis of the first portion of the duodenum was observed. A catheter was inserted into the stomach, but this could not be passed beyond the first portion of the duodenum. In addition there was a loose attachment of the mesentery, with malrotation of the colon, the cecum and appendix being in the right upper quadrant. There was a narrow band extending from the cecum across the second portion of the duodenum, which was divided. An anastomosis was made between the third portion of the duodenum and the distal end of the stomach. Postoperatively the child did quite satisfactorily, and the abdomen became flat and

meconium stools were passed by rectum. Iodized oil given by mouth showed satisfactory progression through the small intestine, and feedings were started on the third postoperative day. However, on the sixth postoperative day the child suddenly became cyanotic, with an episode of extreme respiratory distress, and it was felt that he had massive pulmonary edema or pneumonia. He went downhill quite rapidly and died on Feb. 23, 1949. Postmortem examination showed atresia of the duodenum with a functioning gastroduodenostomy. In addition there was acute pulmonary edema and congestion, congenital malrotation of the colon, congenital hypoplasia of the kidney, and bilateral undescended testes.

T. G., a white boy, born Nov. 20, 1949, birth weight 6 lb. 8 oz. This child was a well developed, well nourished, white boy, who began, when he was about 16 hours old, to have emesis of copious amounts of light green mucus. On the second and third day he was started on skim milk, and he would retain these feedings for two or three hours and then regurgitate in projective fashion. A diagnosis of bowel obstruction was made, and he was taken to surgery, where a congenital stenosis of the second portion of the duodenum was observed. There was, in addition, a malrotation and failure of fixation of the mesentery of the small intestine with a volvulus of the entire small bowel. This was replaced and a duodenojejunostomy performed. The initial postoperative course was quite satisfactory, and the child was gradually started on feedings, which were increased. By the 14th postoperative day it was noted that the infant had become slightly jaundiced but was having stools. However, a few days later he vomited and had only two stools, and the following morning it was observed that the abdomen had become markedly distended with board-like rigidity, and the child had coffee-ground emesis. He was again taken to surgery on Dec. 19, at which time a volvulus of the entire small bowel had again occurred, and it was observed that the entire small bowel was gangrenous. The entire area was resected and an anastomosis made between the jejunum, about 10 cm. below the previous anastomosis, to the terminal ileum about 6 cm. above the cecum. Postoperatively the child did surprisingly well initially but gradually lost weight, with multiple frequent stools, and all attempts to support the child gradually failed, and he died on Feb. 1, 1950, at the 69th hospital day. Postmortem examination showed the entire length of functioning small bowel to be about 9 to 11 cm. There was, in addition, duodenal atresia, functioning side-to-side duodenojejunostomy, functioning end-to-end jejunoileostomy, hypostatic pneumonia, bilateral pleural effusion, and obstructive jaundice.

L. F., a white girl, born Oct. 17, 1950, with a birth weight of 6 lb. This child did fairly well for the first few days of life but then began to have recurrent vomiting and inability to retain the feedings and showed weight loss. She was admitted to the hospital on Oct. 28, 1950, when she was 11 days of age, and x-ray examination revealed a high-grade obstruction of the third portion of the duodenum. The child was prepared with parenteral fluids for a few days, and on Nov. 3, 1950, exploration was carried out, at which time a congenital stenosis of the third portion of the duodenum was observed. A duodenojejunostomy was performed, after which the child made a fairly satisfactory convalescence and was dismissed from the hospital on Dec. 19, 1950, in a satisfactory condition.

S. S., a white girl, born after an uneventful pregnancy on Dec. 1, 1953. This child in the first few days developed symptoms of upper intestinal obstruction with vomiting and repeated regurgitation. The diagnosis of congenital stenosis of the duodenum was made, and exploration was carried out on Dec. 6. At this time a stenosis of the second portion of the duodenum was observed, and a duodenojejunostomy was performed. This child made a very satisfactory convalescence and was dismissed from the hospital on the ninth postoperative day and has since been followed by her local physician.

M. W., a white girl, born Oct. 7, 1950. This child had typical findings of atresia of the esophagus without a tracheoesophageal fistula. X-ray examination revealed no gas in the abdomen, and a catheter could not be passed through the esophagus. On Oct. 10 exploratory laparotomy was done, at which time it was observed that the upper portion of the esophagus was exceedingly short, and anastomosis in a primary fashion would be impossible. For that reason a gastrostomy was performed, after which the child did fairly satisfactorily. On Oct. 18 cervical esophagostomy was performed, and the child again did fairly well until it was observed that considerable difficulty was being encountered through the gastrostomy feedings, and a study with iodized oil revealed high-grade obstruction in the duodenum. Exploration was again done, on Oct. 25, at which time a stenosis of the third portion of the duodenum was observed and a gastroenterostomy was performed. After this the child made a satisfactory convalescence. The child continued to do well and has since had a reconstruction of the esophagus done by a retrosternal jejunal loop, the first stage of which was done on June 25, 1952, with subsequent reimplantation of the lower portion of the jejunum on the stomach on Nov. 26, 1952. This child was last seen on June 4, 1954, in excellent condition, eating normally and developing as a normal 4-year-old child.

J. R., a white boy, born Jan. 5, 1947, with a birth weight of 6 lb. 4 oz. This child very soon developed signs of intestinal obstruction and became markedly distended. X-ray examination revealed typical findings of intestinal obstruction, and exploration was carried out on Jan. 9, 1947. At the time of exploration an area of atresia was observed in the jejunum, which was about 2 in. in length. An enteroanastomosis was made about the area of atresia, but the child continued to have considerable difficulty in the convalescent period with recurrent obstruction. On the fifth day postoperatively complete wound separation was observed, and the child was again taken to the operating room, on Jan. 14, 1947. At this time the previous anastomosis was inspected, and it did not appear to be functioning; so the area of atresia was completely excised, the two ends of the jejunum inverted, and a side-to-side jejunojejunostomy carried out. After this the child made a slow convalescence and was dismissed from the hospital on Feb. 21, 1947. He was seen several times after this and continued to have bouts of chronic small bowel obstruction, and on many occasions visible peristalsis could be observed through the thin abdominal wall. The child was then transferred to the University Hospital, and in June, 1948, exploration was again done and an extensive enterolysis was carried out, and the blind end of the side-to-side anastomosis was resected. After this the child improved, and a telephone call to the patient's mother on Oct. 1, 1954, was made, at which time she stated that the child was entirely normal and was going to school and getting on with no further difficulty.

G. S., a white girl, born April 24, 1953. This child was one month premature and at birth weighed 3 lb. 6 oz. On the first day it was observed that she regurgitated the feedings, along with much greenish-stained mucus. X-ray examination of the abdomen was made, which showed multiple large loops of dilated bowel, and, in addition, a perforation in the colon was observed when an iodized oil enema was given. On April 27 exploration was carried out, at which time multiple areas of atresia were observed in the ileum, and an anastomosis was made between the midjejunum and cecum. In addition, a longitudinal tear in the rectum was sutured, which had undoubtedly been caused by previous instrumentation. The patient tolerated the surgery fairly well but did not ever completely recover and died about 20 hours postoperatively. The essential findings at autopsy were prematurity, multiple atresias of the ileum, bilateral atelectasis, recent jejunocecostomy, and hypertrophy and dilatation of the jejunum.

S. R., a white boy, born March 15, 1952. This child's mother was a diabetic but carried the child to delivery without difficulty and the birth weight was 6 lb. 5 oz. The child was first fed on the second day, but he immediately regurgitated all his feeding, and thereafter the abdomen became distended. The abdominal distention increased, and the child was taken to surgery; the jejunum was found to be greatly dilated and ended in a blind pouch. There was no intervening bowel between that point and the ascending colon, and no cecum was present. The length of the jejunum measured 29 cm. The colon was about the size of a lead pencil but appeared to be normal. An end-to-end anastomosis was made between the colon and the blind end of the small bowel, and the postoperative status appeared to be fairly satisfactory. The child was treated with continuous nasal suction, and he had several stools. However, he subsequently developed fecal emesis, and repeated x-ray examination showed no evidence of gas progressing through the colon. The distention was recurrent, but finally obstruction was apparently again complete, and on April 28 reoperation was carried out, at which time the anastomosis was found to be patent but was somewhat kinked and not functioning. The bowel was aspirated, and a rectal tube was inserted from below through the anastomosis. On the second postoperative day the child developed signs of severe cardiorespiratory collapse, and he died on April 30, 1952, on the 47th day. Postmortem examination revealed congenital absence of the ileum and cecum, jejunocolostomy with partial atresia, and atelectasis of the lungs.

S. O., a white girl, born Jan. 4, 1945, admitted to the Childrens Hospital March 1, 1945. The presenting complaint was vomiting and distention, present since birth. X-rays showed enormous megacolon with narrowing and stricture of the rectum and sigmoid. On March 6, 1945, a cecostomy was performed. However, the bowel content was packed so solid that adequate decompression could not be carried out at the time of operation. The cecostomy was subsequently opened, and after some time the bowel content was evacuated. Because this fecal fistula did not entirely divert the bowel content, a left inguinal colostomy was performed on March 30, 1945. The patient was in critical condition for several months, requiring parenteral fluids most of the time. Subsequently numerous dilatations of the stenotic cecostomy were carried out. On Nov. 23, 1948, a right colectomy by means of obstructive resection was performed, together with closure of an ileocolic fistula that apparently had developed after the first operation. The patient's general condition improved, but there seemed to be some chronic small bowel obstruction. The double-barrelled ileostomy-colostomy was closed by resection and end-to-end anastomosis Feb. 28, 1949. Because of dysfunction of the bowel due to atony of the ileum and distention of the colon, a resection of a segment of dilated ileum and colon was carried out April 10, 1950. On Oct. 12, 1950, the left sigmoid colostomy

was closed. This patient got along fairly well but had bouts of bowel obstruction intermittently. On Feb. 11, 1952, a left colectomy was carried out, together with resection of a segment of terminal ileum. The ileum was then anastomosed to the sigmoid colon. The patient did fairly well but again had intermittent obstruction. On Oct. 13, 1952, the remaining portion of the sigmoid was resected, together with some more of the terminal ileum. At this time a closure of a large defect in the mesentery at the site of former anastomosis was closed. An end-to-end anastomosis was performed between the ileum and the rectum. Since that time the patient has gotten along well and has had no further trouble with obstruction.

Results.—The survival rate in this small series of cases was 62%. This compares favorably with other reported series † but still leaves room for improvement. Because of the association of other anomalies and prematurity, a substantial mortality will probably always exist. The unfortunate significant occurrence of mongolism in the cases of duodenal atresia has been mentioned by Gross,[7] and one of our survivors unhappily substantiates this observation. The causes of death in this series will be noted in the case histories. It is hoped that with earlier diagnosis, improved anesthesia, and more diligent preoperative and postoperative care an increase in survival rate will occur.

VOLVULUS OF SMALL INTESTINE AND MALROTATION OF COLON

In these conditions also the embryological origin has been well established, and to Ladd ‡ must go a great deal of credit for his contributions in diagnosis, surgical management, and improvements in mortality rates for this developmental arrest. The majority of these cases occur within the first few days of life and may be indistinguishable clinically from the preceding group of cases of atresia.

The differential diagnosis is again of academic interest only, as surgical exploration is nearly always indicated in the early case. However, there is also a more chronic recurrent type of symptom-complex which may occur in the older age group and, as will be noted in the case histories, one of our patients was a 10-year-old boy.

In the infants a flat plate of the abdomen is again all that is usually necessary to establish the diagnosis of obstruction. In the older group with recurrent attacks, a barium enema may demonstrate the malrotation of the colon, and barium by mouth will show the evidences of duodenal obstruction.

The treatment of these conditions has been well established and we have nothing to add. Again, complete evisceration of the intestinal tract is essential to making an accurate diagnosis and carrying out adequate therapy. The importance of careful preoperative and postoperative management again cannot be overemphasized.

D. G., white boy, born Nov. 3, 1948, after a normal delivery, with a birth weight of 6 lb. 14 oz. The day after birth it was observed that the abdomen was markedly distended and that during the first 24 hours the child had had only one small mucoid stool. Examination revealed the abdomen to be markedly distended with gas, and no palpable masses could be felt. The impression was intestinal obstruction of a congenital origin, and on Nov. 4, about 48 hours after birth, exploratory laparotomy was carried out. A loop of small intestine was found to be distended and gangrenous as a result of a volvulus, and this loop, together with the cecum and a portion of the ascending colon, were resected and an end-to-end anastomosis carried out between the small bowel and the colon. The postoperative course was satisfactory initially, although the child experienced episodes of recurrent distention. However, barium given by mouth and also by barium enema showed the anastomosis to be functioning satisfactorily. The child, however, soon contracted an infectious diarrhea, from which he never recovered in spite of all attempts at supportive therapy, and he eventually died on the 97th hospital day. The postmortem findings showed the length of the bowel from the pylorus to the anastomosis to be 22 in. and from the anastomosis to the anus 18 in. The additional findings were atelectasis of the lungs, patent ductus arteriosus, peritoneal adhesions.

T. S., white boy, born Feb. 13, 1949, birth weight 5 lb. 14 oz. This child was born at another hospital but apparently shortly after birth the infant started to vomit bile-stained material, and at about the age of 2 weeks an exploratory laparotomy was done. At this operation, according to the mother, there was found "an ulcer opening into the belly with milk escaping into the abdomen." No pyloric tumor was

† References 2, 6, 7, 12, and 14.

‡ References 9 and 10.

found. Since the operation the child had occasional chocolate-colored emeses and stools streaked with fresh and old blood. For two weeks prior to admission the child had some signs of obstruction, and on the day of admission had vomited all of his feedings and had been having bloody emeses and stools. Examination revealed a 12½-week-old white infant, who was quite malnourished, wasted, and chronically ill in appearance. X-ray examination of the gastrointestinal tract was done by means of iodized oil injected in the stomach, and partial obstruction of the second portion of the duodenum was observed, which was thought possibly due to stricture or congenital bands. In addition there was a nonrotation of the colon, which was located in the left upper abdomen. The child was placed on continuous gastric suction and hydrated parenterally for several days. On May 18, 1949, exploration was carried out, at which time numerous adhesions were found in the region of the old incision and liver. The duodenum was found to be constricted by a peritoneal band, which was divided, extending from the malrotated colon across to the right posterior lateral wall of the abdomen. No other evidence of pathology was observed, and the abdomen was closed. Postoperatively the infant's condition remained fairly satisfactory and the abdomen was flat, although he continued to have occasional loose, black stools. On May 31, about two weeks postoperatively, this child suddenly began to gasp fitfully and died quite suddenly. Postmortem examination revealed a large peptic ulcer of the esophagus with ulcerating esophagitis, postoperative intestinal adhesions, and inanition.

J. W., a white boy, born March 16, 1950, with birth weight 7 lb. 12½ oz. Shortly after birth it was observed that the child's abdomen was distended and generally edematous, and the child was spitting up meconium. He was transferred to the Childrens Hospital, at which time generalized edema was noted, and the abdomen was distended and rigid. A surgeon was called in consultation, who explored the baby, and apparently at the time of surgery the child's condition was quite unsatisfactory. The abdomen contained much bloody fluid, and the bowel appeared grayish in color. A drain was inserted, and the abdomen was closed. After this, however, the child continued to respond quite satisfactorily, although the distention and rigidity remained. X-ray examination was made on March 21, 1950, with iodized oil injected into the stomach, at which time upper small bowel obstruction was observed. After repeated transfusions and intravenous fluids, exploration was again done, on March 21, 1950. The old incision was opened and universal adhesions of the bowel to the abdominal wall were found. A necrotic segment of small bowel was observed, with a volvulus noted with a 360 degree twist at the base. An enterolysis of the entire small bowel was carried out and an end-to-end anastomosis made in the jejunum, with an interrupted single-layer suture. Microscopic section of the removed specimen showed it to be ulcerating gangrenous bowel. Postoperatively the child's convalescence was gratifyingly smooth, and on March 26 x-rays showed no evidence of bowel obstruction. The child was dismissed from the hospital on April 26, 1950, weighing 6 lb. 4½ oz. He was last seen on Oct. 22, 1951, with a slight discharge from the umbilicus, which was cauterized, but in other respects the child was normal and developing satisfactorily.

N. T., white girl, born Nov. 15, 1950, with a birth weight of 4 lb. 15 oz. This child showed symptoms of intestinal obstruction, with vomiting and distention shortly after birth, and was admitted to the Childrens Hospital on Nov. 18. After preoperative preparation on November 19, exploratory laparotomy was carried out, at which time a 360 degree rotation of the entire small bowel was found about the superior mesenteric vessels. This was corrected without difficulty, and no strangulation was present. A catheter was inserted through a gastrotomy opening in the anterior wall of the stomach and could be passed well into the duodenum and proximal jejunum without difficulty. The child made an uneventful convalescence after this procedure and was dismissed from the hospital on Dec. 3, 1950, entirely asymptomatic.

R. G., white boy, aged 10 years. This child was first seen Aug. 12, 1948, giving a history of recurrent abdominal pain with vomiting since early childhood. These bouts were recurrent in nature and ordinarily would be relieved by vomiting. X-ray examination was done, which showed malrotation of the colon and upper intestinal obstruction. On Aug. 24, 1948, exploration was carried out, at which time it was observed there was a malrotation of the colon and small bowel, with the cecum lying in the left upper quadrant. The stomach and first portion of the duodenum were enormously dilated, and there were many adhesions which ran anteriorly across the duodenal surface. The adhesions were divided, but there appeared in addition to be marked obstruction of the duodenum by the superior mesenteric artery, and for that reason a duodenojejunostomy was done about the obstruction at the superior mesenteric vessels. The child made a very satisfactory convalescence and was dismissed from the hospital on Sept. 9, 1948. He was last seen Sept. 13, 1948, at which time he was completely recovered and had had no further attacks of obstruction.

Results.—Again the survival rate in these few cases was 60%. As will be noted in the case history, one of our failures seemed to be an early technical success but the child eventually succumbed to an uncontrollable

infectious diarrhea. However, another of our cases is most gratifying, as at the initial operative procedure the prognosis appeared hopeless and nothing definite was done. A secondary exploration, carried out a few days later, at which time a rather extensive resection was done, resulted in complete recovery, demonstrating that even the most desperate cases can occasionally be salvaged. It is again hoped that with early diagnosis and skillful surgical management an improved survival rate can be accomplished.

MECONIUM ILEUS

Meconium ileus has proved to be one of the most difficult obstructive mechanisms to deal with and is still followed by a formidable mortality. This is not surprising in view of the multiple areas of pathologic involvement which make up the entire picture. Many cases which may survive the obstructive phase initially will eventually succumb to the disease because of the pancreatic and pulmonary pathology.

The differential diagnosis here is of considerable importance, as an occasional case may be salvaged by medical measures. Palpation of the firm abdominal masses and x-ray evidence of areas of calcification and granular-appearing intestinal loops will suggest the proper diagnosis in the majority of cases.

A wide variety of surgical procedures have been attempted and none of them have been uniformly successful. Both of our cases were treated by ileotomy and irrigation and expression of the obstructive meconium. There was one survival.

We have had no experience with the Mickulicz exteriorization procedure recommended by Gross [7] but may employ it in the future.

It would seem that improvement in medical and dietary management as well as better surgical techniques will be necessary for a reduction in the mortality of this discouraging condition.

J. M., white boy, born July 15, 1948, with a birth weight of 6 lb. 10 oz. Shortly after birth it was observed that the child's abdomen became increasingly distended with persistent bile-stained vomitus. X-ray examination showed a high-grade small bowel obstruction, which was unimproved after 12 hours of gastric suction. The child was transferred to the Childrens Hospital, where a diagnosis of bowel obstruction on the basis of meconium ileus or possible volvulus was made. On July 17 exploration was carried out, at which time solid meconium was observed to be blocking the small bowel from the first portion of the jejunum to the ileocecal valve. An attempt was made to decompress the bowel by insertion of a needle, which was unsatisfactory; afterward a No. 14 catheter was inserted, and it was again impossible to aspirate any of the tenacious meconium. A transverse incision was made in the terminal ileum, after which a large quantity of saline was injected and the small bowel was milked free of the meconium as completely as possible. The defect in the ileum was then closed with interrupted nonabsorbable (silk) sutures and the abdomen closed. The child made a satisfactory convalescence after this procedure and was dismissed from the hospital in satisfactory condition. The boy was last seen on April 23, 1951, at which time he looked in a very satisfactory condition and had had no respiratory infections.

M. L., white boy, born May 17, 1949. This child was born after a normal delivery, but since birth the abdomen had been observed to become progressively distended, with no bowel movements. There was a family history of a 4-year-old sibling with congenital pancreatic fibrosis. Physical examination revealed a one-day-old white boy with a markedly distended abdomen. An iodized oil enema was given, which filled the entire colon, and it was observed that the terminal ileum contained fairly large masses of rather foamy-appearing material which suggested a meconium ileus. The child was placed on gastric suction without improvement, and on May 20 exploratory laparotomy was carried out. The terminal ileum was packed solidly with hard inspissated meconium, and a small transverse incision was made, after which a large amount of meconium could be evacuated. About 30 cc. of pancreatic extract was injected into the terminal ileum before the enterotomy opening was closed. Postoperatively the child did fairly well for the first few days, and the suction was discontinued on the third postoperative day. The child began to have firm stools about 10 days postoperatively, although he occasionally regurgitated his feedings. On the 15th postoperative day he was observed to have a rather profuse diarrhea and became quite dehydrated. The abdomen again became distended, and the child died suddenly on June 5, 1949. Postmortem examination revealed fibrocystic disease of the pancreas, pulmonary congestion with bronchopneumonia, and chronic low-grade peritonitis.

CONGENITAL INTUSSUSCEPTION

This case of congenital intussusception [11] is included because after a preliminary survey of the literature it would appear to be exceedingly uncommon. In Gross's [7] series of 702 cases, the youngest patient was 3 days of age. Clubbe,[1] reporting on 270 cases, states his youngest patient was 3 months old. Perrin and Lindsay [13] in their monograph based on 400 cases, state their youngest patient was one day old. Hogg and Donovan [8] report on 123 cases and state that one patient was less than one month of age.

Because of its apparent rarity the following case is reported.

D. D., white boy, born on March 1, 1943, with a birth weight of 6 lbs. 4 oz. Examination at the time of delivery showed a tear in the right scrotum with a large scrotal hernia on the left. The penis could not be seen because of the edema of the prepuce and scrotum. The child was taken to surgery about 12 hours after delivery. At the time of surgery it was observed that the right scrotum had a long laceration, about 4 in. in extent, which communicated with an opening in the hernial sac from which bile-stained fluid escaped. The prepuce was retracted, a catheter was inserted into the bladder, and a few cubic centimeters of clear urine was obtained. The hernia was repaired by ligation of the sac, and a similar incision was then made on the left side and a large hernial sac was opened there, and again some meconium-flecked fluid was obtained from the general peritoneal cavity. The hernial sac was ligated and both wounds closed with interrupted nonabsorbable (silk) sutures. A low left rectus incision was then made, and on opening the abdomen we encountered a considerable amount of bile-stained fluid and meconium. A search was made for the perforation in the bowel, and this was found where an intussusception had occurred in utero and apparently was several weeks in duration. A segment of intussuscepted bowel could not be reduced, and about 1½ in. of intussuscepted bowel, which appeared to be mummified, was resected. It was determined then that the bowel at the point of intussusception had been lacerated nearly across the entire lumen, allowing the escape of the bile and intestinal contents to distend the abdomen and thus push out the hernial sacs, which had then become ruptured during delivery. An end-to-end anastomosis was carried out. Four grams of sulfanilamide powder was left about the anastomosis and closure carried out with interrupted absorbable (gut) sutures. The following day, March 2, because of marked edema of the prepuce, a dorsal slit was carried out and a soft No. 8 catheter passed into the bladder. Postoperatively intermittent gastric aspirations were carried out and some old bloody clots were observed to be passed on about the sixth day, but this passage subsequently stopped. Feedings were started on the ninth postoperative day, and the infant was discharged from the hospital on April 9, 1943, on the 39th hospital day, with a weight of 6 lb. 11¾ oz. The child was last seen on Dec. 23, 1947, at which time the abdomen was excellent and both testes were in the scrotum. The child was otherwise normal.

SUMMARY AND CONCLUSIONS (TABLE 2)

Atresia and other obstructions of the intestine occur more frequently than is generally realized.

Early diagnosis and improvements in preoperative and postoperative care and in anesthesia have improved the survival rate considerably.

TABLE 2.—*Summary of All Patients*

Type and Location	No.	Living	Dead
Atresia of duodenum	7	5	2
Stenosis of duodenum	5	3	2
Atresia of jejunum	2	1	1
Absence of ileum and cecum	1	0	1
Stenosis of colon	1	1	0
Volvulus of small bowel and malrotation of colon	5	3	2
Meconium ileus	2	1	1
Congenital intussusception	1	1	0
	24	15 (62%)	9

Although a significant percentage of these children will always be lost because of associated incompatible anomalies, a far greater number can be salvaged and be restored to lead entirely normal lives.

DISCUSSION

DR. EARL PARSONS LASHER JR., Seattle: I realize that probably Dr. Lannin's paper was not meant to be all-inclusive, but, to bring up a point which he did not mention and which has come to our attention in recent years in Seattle, I think he might have included congenital megacolon in the causes of obstruction.

Recently we have had three cases, one of whom died with a diagnosis of intestinal obstruction, megacolon not having been considered as a possible diagnosis. I wonder if he has found any in this series.

DR. B. G. LANNIN, St. Paul: We have had cases of congenital megacolon, but we did not include them in this paper.

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Diverticulitis

Progress Toward Wider Application of Single-Stage Resection

EDWARD S. JUDD Jr., M.D.
and
THOMAS W. MEARS, M.D., Rochester, Minn.

In the past the majority of patients suffering from diverticulitis have been treated by the internist. Only about a fifth of these patients have been sent for surgical treatment. Very properly the treatment at the time of operation has been limited to management of the complications of the underlying diverticulitis. Because of the nature of the lesion, sound surgical principles based on extensive experience over a long period have dictated that the patient must undergo a multiple-stage operation. In most instances it was proved that the attempt to excise the inflamed portion of bowel and reestablish continuity all in one operation was indeed foolhardy. In more recent years the dramatic improvements which have come about in the field of intestinal surgery have stimulated certain surgical leaders to consider single-stage operations in isolated instances of diverticulitis. We have recently had occasion to test out the newer principles and have been very well satisfied with the ultimate results.

Babcock,[1] in 1941, made an early plea for extending operative treatment of diverticulitis to a greatly increased percentage of patients. He wrote:

> Diverticulitis of the sigmoid is the one disease of an intra-abdominal organ characterized by a tendency to persist or relapse with perforation, peritonitis, abscess, fistula, or intestinal obstruction for which leading abdominal surgeons still advocate medical measures and delayed and palliative operations.

Soon other surgeons began to make known their dissatisfaction with the prevailing methods of surgical treatment for this condition. In 1945, Rosser[2] stated that the prevailing methods were unsatisfactory and he advocated earlier operation for many of these patients. This view was quite in contrast to one which he had voiced under similar circumstances some five years previously.[3] In 1945, Martin and Adsit[4] suggested that improved results might be realized if patients in impending danger could be recognized and sent earlier for surgical treatment. In 1948, Herrmann[5] also made a vigorous plea for earlier surgical therapy. In that same year, Babington,[6] reiterating this plea, suggested that all diverticula be resected as soon as they became inflamed. In 1950, Boyden[7] pointed out that multiple-stage operations were not as necessary as had been presumed previously and he was convinced that the incidence of one-stage resections should rise steadily.

Since 1950 surgeons have shown more and more interest in early resection for certain patients suffering from diverticulitis of the sigmoid colon. As the morbidity and the mortality rates associated with intestinal resection have been lowered progressively by the striking improvements in our methods of anesthesia and preoperative and postoperative care, not the least of which is the dramatic development of antibiotic therapy, certain patients with recurring diverticulitis might now be considered candidates for elective interval resection. This statement can now be made in spite of a very definite stand taken by Pemberton, Black, and Maino

The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Section of Surgery, Mayo Clinic and Mayo Foundation (Dr. Judd). Fellow in Surgery, Mayo Foundation (Dr. Mears).

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at the Mayo Clinic in 1947 [5] when they reported experiences with the surgical treatment of diverticulitis extending over the period from 1908 to 1945. These authors emphasized that resection of the involved segment of colon is the only proper definitive surgical treatment for diverticulitis. Only a third of the patients in their series remained well after establishment of a colonic stoma proximal to the involved segment of colon and subsequent closure of the stoma without resection of the involved segment. It was apparent from their review that the treatment of choice for the vast majority of patients was multiple-stage resection; in many cases two colostomies resulted and the patient, of necessity, was resigned to a long period of inactivity which often lasted more than a year, until the various stomas were closed and he was permitted to resume his normal activities.

In recent years, because of changing views on the subject and our earlier enthusiasm after some gratifying results with isolated instances of one-stage resection, we have seriously considered adopting a formal program of elective interval resection for patients suffering from recurring bouts of diverticulitis. To fortify our argument with facts, we were stimulated to review the records of patients having diverticulitis who underwent one-stage resection with primary end-to-end anastomosis at the clinic. Although we were looking especially for the type of case in which the operative procedure might be termed a "prophylactic resection," we realized that, in order to obtain all pertinent data, we should include the case of any patient who underwent one-stage operation regardless of the surgeon's reason for deciding to perform this operation.

MATERIALS AND METHOD

In order to eliminate cases studied prior to the time when modern adjuncts to surgical therapy were available, we selected a period starting in 1946. This coincided also with the termination of the study reported previously by Pemberton, Maino, and Black.[5] Since a prolonged follow-up study was not necessary as we have definite information on long-term results, recurrence rates, and so forth, we continued the study through the year 1953. Of the 329 patients undergoing surgical treatment for diverticulitis in that period we found that 68 had been treated by single-stage resection with primary end-to-end anastomosis. It was of striking interest to us to compare the difference in incidence of one-stage resection in the first four-year period of our study with that in the second four-year period. This variation in numbers represents a trend in present-day treatment (Table 1). The surgeon should be the first to admit that the improvements have not been of his own doing but rather have reflected the cooperate enterprise of the chemists, the bacteriologists, and the internists. Surgical technique has not varied in any striking degree but surgical thinking has altered considerably because of the improved conditions under which the present-day surgeon can work.

TABLE 1.—*Number of Single-Stage Resections Performed for Diverticulitis: From 1946-1953 (Sixty-Eight Cases)*

Period	Patients	
	No.	Per Cent
1946-1949	15	22
1950-1953	53	78

SEX AND AGE OF PATIENTS

In our series of cases 41 patients were men and 27 were women. The ratio of men to women was 1.5:1. The preponderance of men in the series agrees with the incidence reported by other authors. The average age of our patients was 56.4 years, the range being from 38 to 78 years.

COMPLICATIONS OF DIVERTICULITIS

Since our study included all cases in which patients having diverticulitis underwent one-stage resections in a given interval of time, we, of necessity, included all of the complications of diverticulitis, and in some instances more than one complication was exhibited by the same patient. Strictly speaking, some of the patients were operated

on for uncomplicated diverticulitis and these features will be elaborated later. Only 15 of the patients were found to be free from all complications at the time of resection. The preoperative complications of diverticulitis which were noted most frequently were abdominal mass, obstruction, hemorrhage, and fistula.

Abdominal Mass.—Masses were palpable in 21 patients: in 13 patients masses were detected on digital examination of the rectum, in 6 they were palpable bimanually, and in 2 they were palpable only by abdominal examination. It is interesting that two patients underwent surgical exploration with a diagnosis of ovarian mass. Even when the correct diagnosis became evident later, we were still unable to obtain a history of symptoms referable to the colon. This merely emphasizes that one cannot always predict what stage of the disease process is present by mere study of the severity of symptoms.

Obstruction.—Twenty patients had signs and symptoms of obstructive phenomena in the sigmoid colon. Only two patients, however, revealed complete obstruction to the retrograde flow of barium during fluoroscopic study. We found that when obstruction was a feature, it was usually a result of repeated episodes of inflammation and, therefore, characteristically chronic and incomplete.

Hemorrhage.—The recent surgical literature tends to stress hemorrhage more and more as an indication for resection in diverticulitis. Although the origin of colonic bleeding usually cannot be demonstrated in many cases in which diverticulitis is implicated as the primary source of bleeding, it is noticed that hemorrhage is included in most reports as a predominant factor. In our series definite efforts were made to eliminate such causes as hemorrhoids, polyps, benign ulcerations, malignant ulcerations, and hemangiomas before assuming that the bleeding was due to diverticulitis alone. In six patients, however, in whom bleeding had been an alarming symptom, no other cause could be demonstrated aside from diverticulitis. It so happened that these patients had other indications for surgical intervention, and in the final analysis it developed that none of our patients underwent resection with hemorrhage as the only indication.

Fistula.—Fifteen patients were found to have fistulas as a complication of their diverticulitis. The commonest site for the fistulas was between the sigmoid colon and the urinary bladder and for obvious reasons they occurred predominantly among the men. Twelve of the 15 patients proved to have sigmoidovesical fistulas, and 10 of the 15 were men. The other three patients had the following types of fistulas: one had a sigmoidoileovesical, one had a sigmoidovaginal, and one had a sigmoidocutaneous fistula.

TABLE 2.—*Indications for Single-Stage Resection in Sixty-Eight Cases of Diverticulitis*

Condition	No. of Cases
Question of malignancy	18
Recurring attacks of diverticulitis	17
Formation of fistula	15
Extrarectal mass	11
Obstruction	4
Incidental operative finding	2
Persistent severe diverticulitis	1
Total	68

INDICATIONS FOR SURGICAL TREATMENT

We carefully analyzed our series to determine just what the indication might have been for single-stage resection with primary end-to-end anastomosis (Table 2). We found that the indications in our series were somewhat similar to those found by others. Seventeen patients presented themselves because of recurring attacks of diverticulitis. Of these 17, two gave histories of extreme frequency of urination during each attack. Since both of these patients were men, it seemed extremely important to us to consider this as nature's warning that a vesical fistula was imminent. Four other patients of this same group also presented histories of recurring bouts of colonic obstruction which occurred whenever the inflammatory episode was at its height. Of the 68 patients in our entire series, these 17 aroused our interest most,

since it appeared to us that definite progress was possible in the treatment of these patients. Whereas previously, conservative management of these patients was common practice, we are now convinced that, with the mortality and morbidity figures being what they are today, we have a great deal to offer these patients when we see them during the inactive interval of their diverticulitis if we stress the advantages of elective, orderly resection.

The two patients who underwent resection of the sigmoid because of an incidental operative finding demand a bit of explanation. One patient was operated on under emergency conditions because of an incarcerated abdominal hernia. The discovery of advanced diverticulitis seemed to indicate immediate resection and, fortunately, the patient tolerated this very well. The other patient, prepared for intestinal surgery as a matter of routine, was undergoing an abdominal operation for rectal prolapse. A surprising finding was advanced diverticulitis of the sigmoid and, since the patient was well prepared for intestinal surgery, resection of the bowel was promptly carried out. This procedure not only cured the diverticulitis but also facilitated the operation for the prolapse.

POSTOPERATIVE COMPLICATIONS

The complications which developed after resection for diverticulitis were those which would be expected after intestinal resection for other reasons (Table 3). Only one of the two fecal fistulas seemed to be of any consequence, but this ultimately healed. The patient who suffered a cerebrovascular accident improved steadily while at the clinic but still had some residue in the form of paralysis at the time of his dismissal. The patient with intestinal obstruction became so distended that he experienced dehiscence requiring secondary closure of his wound. Even considering all of the complications listed in Table 3, the average length of hospitalization after operation was found to be 17 days.

The hospital mortality rate in this series of cases seems to be quite acceptable. Although the series is not large, the fact that all but one patient survived seems to represent better than average results. Curiously enough, death of this one patient was due to a condition totally unrelated to his intestinal resection. On the 16th postoperative day he had a sudden exsanguinating hemorrhage from a duodenal ulcer and time did not permit any definitive treatment.

COMMENT

In the past the surgical treatment of diverticulitis has been limited to those patients exhibiting one and frequently more than one complication of the disease. We are of the opinion that a reevaluation of this philosophy is in order. Present-day improvements in anesthesia and preoperative and postoperative care, including the advances in antibiotic therapy, have brought about a radically different situation. The great lowering of the morbidity and the mortality rates in this type of surgery suggests that progress can definitely be made toward offering so-called prophylactic resection for certain patients known to have uncomplicated diverticulitis at the time of examination. In these cases the history is most important. Although the patient may appear to be in excellent health and roentgenograms may reveal evidence of only scattered diverticula in the left portion of the colon, the history of repeated episodes of considerable inflammatory change should be enough to stimulate the conscientious surgeon to do his utmost to avoid serious complications. Especially in the male, these complications can be not only distressing but also incapacitating for one to two years. A single-stage operation can be

TABLE 3.—*Postoperative Complications After Single-Stage Resection for Diverticulitis (Sixty-Eight Cases)*

Complication	No. of Cases
Temporary fecal fistula	2
Minor pelvic abscess	1
Wound infection	1
Cerebrovascular accident	1
Intestinal obstruction	1

carried out under ideal circumstances which will spare the patient the increased risk, discomfort, inconvenience, and expense associated with the multiple-stage procedure which he might well be required to undergo later if the somewhat obsolete policy of watchful waiting is allowed to continue. We are in full accord with those authorities who insist on multiple-stage operations for the complicated cases and we would stress again the sound principles of never taking a chance and forcing the issue. Since one is dealing with a benign process, the over-radical approach of attempting to remove truly inoperable lesions cannot be condoned especially when a period of rest produced by a transverse colostomy will render a vast difference in the operative field.

It now appears to us that there are three groups of patients that we might seriously consider as candidates for elective, prophylactic, single-stage resection carried out under ideal circumstances.

The first group includes those patients who experience recurring attacks of diverticulitis in spite of good medical care. It seems obvious to us that the more repeated attacks of diverticulitis a patient is allowed to experience the more likely it is that a serious complication of his disease will develop. Not only are these attacks most unpleasant for the patient and not only do they inconvenience him but also the economic loss, if the patient is the head of a family, is such as to be a serious consideration. We see no reason why these patients should be forced to continue to endure these repeated episodes. Also included in this category is the patient in whom a severe and persistent form of diverticulitis has developed. We had only one such patient in our series. We now know that under proper conditions this type of lesion may be attacked in a one-stage operation.* At the risk of considerable repetition we must reemphasize that these patients must not undergo resection during an acute attack of diverticulitis. It is only after the inflammatory change has been treated with good general supportive measures, which would include all of the modern adjuncts now available, and during the so-called quiet interval that surgical intervention should be considered.

The second group includes those patients who have urinary symptoms associated with an attack of diverticulitis. For obvious reasons most of these patients will be males. We must interpret the presence of the urinary symptoms during these attacks as warnings that the inflamed sigmoid colon is threatening the urinary bladder. If we wait until gas is passed by way of the urethra or gross feces can be detected in the urine, we will have waited too long.

The third group includes patients who have indications which cannot be proved to be noncancerous preoperatively. It is quite understandable that this group of patients will form a substantial segment of any series.† We found these indications to be the impelling reason for operation in 26.5% of our cases. The roentgenologist was unable to state the exact nature of the lesion in 22% of the patients who were examined roentgenologically by means of a barium enema. In one instance the roentgenologist reported a lesion very suggestive of carcinoma. In fact in 3.2% of our entire series a definite roentgenologic diagnosis of carcinoma had been made although all the lesions later proved to be benign. In one interesting case a lesion was reported as a carcinoma secondarily involving the cecum; at surgical exploration it was found that all inflammatory reaction was due to diverticulitis of the sigmoid. In defense of the roentgenologists, the surgeons should be the first to admit that the difficulty in differentiation is not limited to the preoperative period alone. Frequently the surgeon cannot be certain at the time of surgical exposure what the lesion is and in some instances he will not know until the pathologist renders his report.

SUMMARY AND CONCLUSIONS

The majority of operations for diverticulitis are, of necessity, performed in multiple

*References 9 to 12.

†References 13 and 14.

stages. Our study has revealed, however, that, in carefully selected cases, one-stage resection is not only feasible but also preferable.

Conservative treatment of diverticulitis for a prolonged period, in spite of repeated, severe, acute attacks, may lead to extreme difficulty from complications.

Prophylactic resection of the colon during an interval when the diverticulitis is quiescent has much to recommend it and has been accomplished with very low morbidity rates and very little risk.

DISCUSSION

Dr. S. B. Harper, Madison, Wis.: My interest in diverticulitis is quite personal as well as professional. My good friend, Dr. Judd, had a purpose when he asked me to comment on his fine paper. The 261st and last case on the chart enumerating the patients undergoing multiple stage procedures represents my own case. Five major procedures, as they are euphemistically called, have admittedly livened my interest in this disease and undoubtedly distorted my surgical judgment.

The trend toward wider application of primary resection with anastomosis in the treatment of colonic diverticulitis such as has been accomplished in colonic neoplasms represents real progress. Proximally diverting or exteriorization procedures in this disease frequently run a hectic course. The total illness period is measured in months instead of days. The adjustment made by the patient to the colostomy, albeit temporary, is a difficult one. A colostomy, in my experience, seems to be more difficult to manage in the presence of diverticulosis and diverticulitis. Elective resection with an infinitely shorter hospital stay and the elimination of a colostomy, in addition, allows the surgeon to resect as much of the diverticulum-infested colon as he deems advisable.

Dr. Judd has outlined very valid indications for applying primary resection and anastomosis in the treatment of diverticulitis. The problem ahead is to make the patient fit the optimal procedure. In the future many more patients will be resected primarily if a little more care and time is provided in preparing the bowel. These inflammatory pelvic masses often respond dramatically to a program utilizing the broad spectrum antibiotics, minimum residue foods, suitable antispasmodic drugs, and sedation-induced bed rest. Perhaps the antiphlogistic properties of corticotropin (ACTH) or cortisone have a place in preparing these patients for a technically feasible primary reaction. Premature exploration of the patient who presents a partly obstructing, hard, tender, pelvic mass may be encouraged by the family or the referring physician, I find. Lately, it appears that a patient extension of the preparatory period for a week or so may shorten the postoperative period by several months. The objection that valuable time is lost, if, instead of an inflammatory lesion, a carcinoma of the ovary or sigmoid is the cause of symptoms, is hardly valid. The resectability of either type of lesion where a "frozen pelvis" is found will be improved under an adequate program such as described.

In the less commonly encountered acutely perforating diverticulitis, providing that it is not confused with appendicitis, a conservative approach can be justified until adequate preparation of the bowel is completed. This is no time to allow administrative pressures of suitable operating hours, family demands, or incomplete control of the patient's care to temper good judgment.

As our medical colleagues could easily tell us, chronic diverticulitis is a commoner disease than the surgeons realize. It would be less easy for any of us to outline what constitutes the so-called medical management. As with the chronic, intractable duodenal ulcer, properly performed resection of the disease-bearing segment of bowel can return these dietary invalids to a life involving only minor abstinences.

Dr. Allen Marston Boyden, Portland, Ore.: I apologize for getting up again so soon; however, this subject has been of great interest to me, and I was particularly delighted to hear Dr. Judd this morning.

In 1948 I reviewed a 20-year experience at the Portland Clinic with the surgical treatment of diverticulitis, and recently have had the opportunity of adding the last five years of our experience. This review has been of interest because of the fact that the early 15 years of this study represented the work of the late Dr. Thomas Joyce, a member of this Association. I thought that you might be interested in what he did and what he taught those of us who followed him.

Actually, in this series of 25 years there were 47 patients who underwent resection of the sigmoid for diverticulitis. Eighty-two per cent of these had resections of the involved colon at the primary operation; 66% had primary resection with anastomosis, and 57% of the total had a primary anastomosis without concomitant colostomy.

There has been one death. This was not one of Dr. Joyce's—it was one of mine. I was particularly amazed at the brilliance of his surgery, the study dating from 1929. I believe from this study that we should reevaluate our whole concept of the surgery of diverticulitis.

Three of these patients, as Dr. Judd mentioned, had one-stage resections for sigmoidovesical fistulae

and were out of the hospital in an average of 14 days after operation. These things can often be accomplished without a long-standing colostomy.

I am interested as well because Dr. Joyce never instituted prolonged colostomy drainage. We have continued to follow his precepts. In the 20% that did not have primary resection, the average duration of colostomy before definitive resection was five weeks. The longest period was 13 weeks, contrary to the usual teaching of from six to nine months.

I am convinced that a colostomy for a benign disease, in old patients particularly, is a morbid condition and should be avoided if possible. The total duration of morbidity in patients who underwent primary resection with anastomosis without colostomy was about one-third as long as those that had a colostomy either with primary resection, preliminary to resection, or a Mikulicz procedure.

I appreciate the opportunity of discussing this interesting paper, and I commend to you more liberal indications for primary resection.

Dr. Foster L. McMillan, Chicago: I thoroughly concur with Dr. Judd's conclusions and the remarks of Dr. Boyden on the extension of the indications for a one-stage resection in the surgical treatment of diverticulitis of the colon.

We can diminish the long periods of disability and the extensive financial burden of many patients by adoption of a one-stage operation procedure in carefully selected cases. The indications for the selection of these cases has been accurately covered, except that I should like to add one other indication: a chronic, distorted, stenotic, or granulomatous diverticulous sigmoid as revealed only roentgenologically. This group of patients should also be taken care of at a selective time by a one-stage procedure.

In addition, I should like to call attention to one other feature which I am sure has not been stressed sufficiently, and that is the coincidence of acute small bowel obstruction occurring with either acute or chronic diverticulitis. An adhesive small bowel obstruction may be overlooked during the course of chronic obstructive diverticulitis. In such an instance a fatal outcome will result even though decompression of the colon has been accomplished by colostomy.

The same applies when obstructive symptoms occur in the acute phase of diverticulitis. The small bowel dilatation, nausea, and vomiting may be erroneously interpreted as ileus secondary to the peritoneal reaction around the perforation resulting from the diverticulitis when actually an adhesive small bowel obstruction may also be present. I have seen three such cases in the last year, in which the small obstruction itself could have been overlooked, and any surgical attempts to palliate the diverticulitis only would have ended disastrously.

Finally, I should like to show very briefly two slides illustrating a case in which massive melena occurred with acute diverticulitis.

A 58-year-old woman known to have had diverticulosis for a number of years had passed small amounts of blood periodically over the past six years. She entered the hospital with massive hemorrhage from the rectum, and this continued. After supportive treatment with transfusions, a barium-in-oil enema was given, revealing an extensive diverticulosis and diverticulitis of the sigmoid colon.

[Slide] The bleeding continued. After preparation by repeated transfusions, antibiotics, and chemotherapy, laparotomy was performed and a primary resection of the left colon was accomplished.

[Slide] The opened resected specimen revealed multiple diverticula of the left colon. Several in the sigmoid were ulcerated at the margins and were filled with blood, obviously the source of the bleeding in this case. For three years this patient has remained well, and there has been no recurrence of bleeding.

Dr. James W. Hendrick, San Antonio, Texas: I have thoroughly enjoyed this comprehensive paper on such a timely medical problem. It is very interesting to note that this phase of abdominal surgery has been neglected by men and groups that have devoted a large amount of their time and effort to abdominal surgery except the cases that developed a surgical emergency, such as acute perforation with abscess, fistula, obstruction, sinuses, and other serious complications.

We have been interested in this phase of abdominal surgery for many years. Since the average age of our population is increasing, diverticulitis of the sigmoid and descending colon is an ever-increasing problem both to the physician and the patient. We have found that 7.5% of the people over 50 years of age that had routine proctoscopic and roentgenological examination of the colon had diverticula. In 20% of this group, symptoms of diverticulitis were present, and in approximately 95% the disease was primarily located in the sigmoid and/or descending colon.

When the disease once definitely manifests itself, the future of that individual case is quite unpredictable and in most instances the progress of the inflammatory change is slow, but such cases may develop rapidly and present serious complications that the surgeon must care for.

In general, the medical profession has adopted a rather ultraconservative or inert attitude toward the surgical therapy of this complicated disease. When palliative measures are continued long after the danger signals have manifested themselves, the condition may progress to a stage where definitive surgery is hazardous and difficult and, in some instances, almost impossible. The reason for this

apathy probably is that the slow progress of the disease in many instances is deceiving. Advanced inflammatory changes may be present in the colon without causing severe symptoms or signs, the physician being unaware that serious complications may occur. Formerly colon surgery had a substantial mortality. In the past, the results of operative therapy were poor, since it was utilized, in most instances, when severe complications occurred, such as perforation, abscess formation, peritonitis, and fistulae. It was generally accepted that the patient was required to have the colostomy for long periods of time before definitive surgery could be utilized to eradicate the diseased area of the bowel. This necessitated multiple operations over a period of months. Fortunately, during the past few years, the more alert members of the medical profession have discarded the ultraconservative attitude toward surgical therapy for diverticulitis and its complications and are now utilizing practical, properly timed, surgical measures with comparatively excellent results. The advent of antibiotics and development of intestinal antiseptics have lessened risks inherent in the resection of the descending or sigmoid colon. This is now accepted as definitive, curative therapy for diverticulitis.

Previously, the trend of thought was that surgical treatment was not indicated unless such complications as unremitting obstruction, perforation, or fistula developed.

During the past few years, we have seen 38 private patients with diverticulitis of sufficient degree to necessitate or to justify surgery, and in 20 of these patients, perforation had developed with abscess formation. Nineteen required drainage of the abscess and colostomy and later resection of the involved area and reestablishment of the continuity of the bowel. In one case, perforation which was not immediately recognized had developed. The patient was treated by her physician with several enemas for 48 hours, during which time general peritonitis developed. The abdomen was filled with enema fluid at the time of surgery and the patient died. Eighteen patients were observed who had severe recurrent attacks of diverticulitis and an increasing progress of the disease which resulted in partial stenosis of the bowel. These patients had resection of the involved segment of sigmoid colon with primary end-to-end anastomosis, permitting them to return to their normal, daily activities with no mortality.

Fortunately, as Dr. Judd has pointed out, the disease is limited to a small segment of the colon, that is, the sigmoid or descending colon. It is suggested that the patients with diverticulosis be constantly followed by their physician. When recurrent attacks of diverticulitis develop that are incapacitating to the patient, he should be considered a candidate for resection of the involved area of the colon with primary end-to-end anastomosis after adequate preparation. In the event that the disease has been permitted to progress to perforation, drainage of the abscess and transverse colostomy and later resection of the involved loop of colon with reestablishment of continuity of the bowel is the procedure of choice.

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The Surgical Treatment of Gastrojejunal Ulceration

WALTMAN WALTERS, M.D.
DONALD P. CHANCE, M.D.
and
JOSEPH BERKSON, M.D., Rochester, Minn.

The first use of vagotomy in the treatment of gastrojejunal ulcer at the Mayo Clinic was in 1945. In the years 1945 to 1952 inclusive, 617 operations were performed on 586 patients for gastrojejunal ulcer. One hundred and forty-three of these operations consisted of vagotomy alone or of vagotomy combined with additional procedures, while 474 were operations in which vagotomy was not done. The latter operations consisted primarily of gastric resection. The 143 patients who underwent vagotomy will be called the "vagotomy group."

A group of 158 patients treated for gastrojejunal ulcer by gastric resection or gastric re-resection during the same interval was studied also and this will be referred to as the "resection group." *

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Section of Surgery (Dr. Walters); Assistant to the Staff (Dr. Chance), and Section of Biometry and Medical Statistics, Mayo Clinic and Mayo Foundation (Dr. Berkson). The Mayo Foundation, in Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

* The "resection group" was selected by listing in ascending order the record numbers of the 474 patients who had operations other than vagotomy and then choosing the records for every third case. If the record chosen indicated that an operation other than resection had been performed, then the record bearing an adjoining number was chosen until we had records of 158 cases of gastrojejunal ulcer in which gastric resection had been performed; these constitute the series included in the present study.

The 301 cases included in this study were divided into two groups: 115 cases in which gastrojejunal ulcer occurred after gastric resection, and 186 cases in which gastrojejunal ulcer occurred after gastroenterostomy. By analyzing these two groups from the standpoint of certain clinical features before and after operation we obtained concrete support for the conclusions of our follow-up study.

CLINICAL ASPECTS

In the 115 cases the mean interval between the partial gastric resection and the subsequent operation for gastrojejunal ulcer was 3.7 years, while in the 186 cases the mean interval between gastroenterostomy and the subsequent operation for gastrojejunal ulcer was 11.2 years.† From these figures it is obvious that the incidence of gastrojejunal ulcer after either gastric resection or gastroenterostomy can be determined only on the basis of a long-term follow-up. This is particularly true after gastroenterostomy and probably explains why the incidence of gastrojejunal ulcer, as first reported, was low but has gradually increased as the time of follow-up has increased.

Gastrojejunal ulcer, like duodenal ulcer, occurs predominantly in men. In the present group of 301 patients, 274 (91%) were men and 27 (9%) were women.

Complications.—The complications of gastrojejunal ulcer are hemorrhage, gastro-

† We recognize that these figures do not indicate the mean interval between the operation and the onset of recurring ulcer-like symptoms. Yet sometimes symptoms of recurring ulcer are not due to ulcer as determined at operation and, as will be shown, there is a high incidence of error in the roentgenologic examination of patients with gastrojejunal ulcer.

jejunocolic fistula, and perforation. Hemorrhage occurs more commonly in gastrojejunal ulcer than in duodenal ulcer, but it is rarely fatal since the vessels in the vicinity of the gastrojejunal anastomosis are usually not large. Among our 301 patients about half (149 patients) gave a history or presented evidence of gastrointestinal hemorrhage on admission. The incidence of hemorrhage from gastrojejunal ulcer following gastroenterostomy was the same as that from ulcer following gastric resection..

Gastrojejunocolic fistula is the most serious complication of gastrojejunal ulcer, and the incidence has been variously reported as 8.7, 11.4 and 13.6% by Judd,[1] Priestley,[2] and one of us (Walters) [3] and associates. Fistula was present in 20 of our 301 cases, an incidence of 6.5%. It occurred mostly as a complication of gastrojejunal ulcer after gastroenterostomy, 16 of our 20 cases being in this group.

Only 2 of the 301 patients were seen with an acute perforation although a number gave a history of acute perforation that had been treated elsewhere.

DIAGNOSIS

The concentration of gastric acids after a modified Ewald meal was determined for 268 of the 301 patients. Free hydrochloric acid was found in 77.6% of the cases in which the gastrojejunal ulcer followed gastric resection and in 95.7% of those in which the ulcer followed gastroenterostomy.

Roentgenologic findings in 275 of our cases indicated a lesion in 79% of the cases in which the gastrojejunal ulcer followed gastric resection and in 66% of cases in which the ulcer followed gastroenterostomy. However, a positive roentgenologic diagnosis of gastrojejunal ulcer was made in only 50% of the cases in which the ulcer followed gastric resection and in 37% of the cases in which it followed gastroenterostomy.

Of 85 cases in which the size of the ulcer crater was given in the operative note, abnormalities had been observed on roentgenologic examination in 64%, but a positive roentgenologic diagnosis of ulcer had been made in only 36%. From this it is evident that the roentgenologist has difficulty in demonstrating a gastrojejunal ulcer and that this difficulty is greater after gastroenterostomy than after gastric resection.

The roentgenologic diagnosis of gastrojejunocolic fistula is made much more easily when barium is given by rectum than when it is given by mouth. In our 20 patients with fistula, 16 had barium enemas and in each the fistula was demonstrated. Of 13 patients who underwent roentgenologic examination of the stomach with the aid of barium, in only 1 was the fistula demonstrated, although 11 had evidence of gastrojejunal ulcer.

OPERATIVE PROCEDURE AND MORTALITY

Gastrojejunal Ulcer After Gastric Resection.—Vagotomy was performed, with one death, in 88 of the 115 cases in which gastrojejunal ulcer followed gastric resection, giving an operative mortality rate of 1.1%. Fourteen patients underwent vagotomy and gastric resection, with two deaths, giving an operative mortality rate of 14.3%. The remaining 13 patients underwent gastric reresection, with two operative deaths, giving a mortality rate of 15.3%. Thus in this group of 115 cases of gastrojejunal ulcer after gastric resection there were five deaths, giving an operative mortality rate of 4.3%. The details of these deaths are summarized in Table 1.

Gastrojejunal Ulcer After Gastroenterostomy.—Of the 186 patients with gastrojejunal ulcer after a gastroenterostomy one (0.5%) died after operation. In 145 of these patients the gastroenteric anastomosis was taken down and partial gastric resection was performed, with one operative death, giving a morality rate of 0.7%; of the remainder, 29 were treated by vagotomy alone, 3 by vagotomy and gastric resection, and 9 by vagotomy and other operations. No deaths followed vagotomy. Included among the patients treated by vagotomy and other operations were three in whom the gastroenteric anastomosis was taken down and pyloroplasty was carried out, two in whom the gastroenteric anastomosis was taken down, and

TABLE 1.—*Deaths in Hospital After Operations for Gastrojejunal Ulcer*

Case	Age, Yr.	Sex	Previous Operation	Symptoms and Signs	Operation	Cause of Death
				Gastrojejunal Ulcer After Gastric Resection		
1	37	M	Two-thirds of stomach removed (elsewhere)	Pain; many gastrointestir ' hemorrhages; hypertension (200/120)	Transthoracic vagotomy	Cerebrovascular accident 39 days after operation
2	45	M	Small part of stomach removed (elsewhere)	Pain; many gastrointestinal hemorrhages	Vagotomy and gastric re-resection	Volvulus of jejunum 14 days after operation; resection, oliguria, and uremia
3	52	M	Small part of stomach removed (elsewhere)	Pain; many gastrointestinal hemorrhages	Vagotomy and gastric re-resection	Acute pancreatitis 17 days after operation
4	57	M	Third of stomach removed (elsewhere)	Pain; many gastrointestinal hemorrhages; active syphilis	Gastric re-resection	Duodenal fistula; subhepatic abscess; gastrointestinal hemorrhage 54 days after operation
5	59	M	Third of stomach removed (elsewhere)	Pain; diarrhea, loss of weight, acute perforation of gastroileal ulcer	Gastric re-resection	Cerebral hemorrhage 17 days after operation
				Gastrojejunal Ulcer After Gastroenterostomy		
6	60	M	Gastroenterostomy (elsewhere)	Pain; one gastrointestinal hemorrhage	Gastric resection	Duodenal fistula, subphrenic and subhepatic abscess 18 days after operation

four in whom a gastrojejunocolic fistula was excised.

RESULTS OF FOLLOW-UP

Gastrojejunal Ulcer After Gastric Resection.—We were able to follow 94 of the 115 patients in this group. Of these, 78 were treated by vagotomy alone with excellent ‡ results in 70.5%, unsatisfactory results in 11.5%, and poor results in 18.0%. Nine patients underwent vagotomy combined with gastric re-resection; all obtained excellent results. Of the seven traced patients who underwent gastric re-resection alone, only 57.1% obtained excellent results.

Gastrojejunal Ulcer After Gastroenterostomy.—Of the 186 patients in this group, 148 were traced. Twenty-seven of these were treated by vagotomy alone, with excellent results in 77.8%, unsatisfactory results in 11.1%, and poor results in 11.1%. Of the two patients that underwent vagotomy and gastric resection, one had an excellent result and one an unsatisfactory result. Of eight traced patients that underwent vagotomy and other operations, 50% obtained excellent results, 25% unsatisfactory results, and 25% poor results.

The remaining 111 traced patients underwent gastric resection; of these, 86.5% had excellent results, 4.5% unsatisfactory results, and 9.0% poor results.

Relief of Hemorrhage.—Of the 124 traced patients in the vagotomy group, 111 (89.6%) had no further hemorrhage.

‡ In our series, 242 patients were followed for periods ranging from one to eight years and the results were classified as follows: (1) excellent, when the patient had no symptoms of ulcer and no evidence of disturbance of motility, such as gastric retention, diarrhea, or bloating; (2) unsatisfactory, when symptoms were suggestive of recurrent ulceration but such ulceration was not proved, or when symptoms of motility disturbance were present, and (3) poor, when recurrent ulceration was proved either by roentgenologic examination, at operation, or both. This is the same classification that one of us (Walters: references 4 and 5) has used in all previously reported studies on vagotomy. The follow-up was based on examinations, laboratory studies, and questionnaires.

Of the 118 patients followed after gastric resection or re-resection, 114 (96.6%) did not have further hemorrhage.

Postoperative Acidity as Indicated by the Insulin Test.—The Hollander insulin test was performed on 71 patients after vagotomy, with negative results in 61 § and positive results in 10. As Walters, Priestley, and Belding have shown,[6] there was uncertain correlation between the results of this test and the final results. Of the 54 traced patients that had negative insulin tests, 69% had excellent results, yet 5 of the 9 traced patients that had positive insulin tests also had excellent results.

Gastric Acidity Before and After Vagotomy and Gastric Resection.—In the vagotomy group the concentration of gastric acid was determined preoperatively for 34 patients who had undergone gastroenterostomy and all 34 had free hydrochloric acid with a mean value of 33.5 degrees (Töpfer's method). Values for acids were determined after operation on 25 of these patients. Seven (28%) were achlorhydric and 18 still had free acid with a mean value of 27.9 degrees.

In the resection group preoperative acidity was determined on 127 patients with gastrojejunal ulcer that followed gastroenterostomy, 7 of whom were found to be achlorhydric, while 120 had free hydrochloric acid with a mean value of 36.4 degrees. Postoperatively 21 of these patients had their acids determined; 18 (85.7%) were achlorhydric while 3 had free hydrochloric acid while a mean value of 15.3 degrees.

Of the 95 patients in the vagotomy group with gastrojejunal ulcer following resection on whom acids were determined preoperatively, 21 (22.1%) were achlorhydric while 74 (77.9%) had free acids with a mean value of 27.5 degrees. Of these, 55 had acids determined postoperatively; 42 (76.4%) were achlorhydric while 13 had free hydrochloric acid with a mean value of 25.5 degrees (Table 2).

§ A "negative Hollander test" was originally thought to indicate complete vagotomy.

TABLE 2.—*Gastric Acidity Before and After Operation for Gastrojejunal Ulcer*

Previous Operation and Time of Test in Relation to It	Acids Determined, Cases	Achlorhydria		Free Hydrochloric Acid	
		Cases	Per Cent	Cases	Per Cent
Vagotomy Group					
Previous gastroenterostomy					
Preoperative	34	..	....	34	100.0
Postoperative	25	7	28.0	18	72.0
Previous gastric resection					
Preoperative	95	21	22.1	74	77.9
Postoperative	55	42	76.4	13	23.6
Gastric Resection Group					
Previous gastroenterostomy					
Preoperative	127	7	5.5	120	94.5
Postoperative	21	18	85.7	3	14.3
Previous gastric resection					
Preoperative	12	3	25.0	9	75.0
Postoperative	4	3	75.0	1	25.0
Total Group					
Previous gastroenterostomy, preoperative	161	7	4.3	154	95.7
Previous gastric resection, preoperative	107	24	22.4	83	77.6

Of those who had a gastrojejunal ulcer after gastric resection and who underwent re-resection, 12 had acids evaluated preoperatively; 3 were achlorhydric and 9 had free hydrochloric acid with a mean value of 36.6 degrees. Four of these patients had acids determined postoperatively; three (75%) were achlorhydric and one had free acids.

Vagotomy by Transthoracic and Transabdominal Approaches.—Of the patients in the vagotomy group 117 had vagotomy alone; 67 of these operations were done by the transthoracic route and 50 by the transabdominal. Of 63 patients followed after transthoracic vagotomy 71% had excellent results, while of the 42 followed after transabdominal vagotomy 74% had excellent results.

Gastrojejunocolic Fistula.—The results of treatment of 20 patients with gastrojejunocolic fistula were good (Table 3). Sixteen of the fistulas followed gastroenterostomy and four followed gastric resection. There were no operative deaths.

Twelve fistulas that developed after gastroenterostomy were treated by excision of the fistulas with closure of the openings in the colon and jejunum and partial gastric resection. Eight of these patients were followed; seven had excellent results and one a poor result due to a recurrent gastrojejunal ulcer. The remaining four patients whose fistula followed gastroenterostomy underwent excision of the fistula, closure of the opening in the colon and jejunum, and vagotomy. Three had excellent results, and one had an unsatisfactory result because of diarrhea subsequent to vagotomy; because of gastrointestinal hemorrhage this patient was suspected of having further ulceration.

TABLE 3.—*Results of Surgical Treatment in Twenty Cases of Gastrojejunocolic Fistula*

Surgical Treatment	Patients Operated on	Patients Followed	Results		
			Excellent	Unsatisfactory	Poor
Previous Operation: Gastroenterostomy					
Fistula excised, openings in colon and jejunum closed plus:					
Partial gastric resection.....	12	8	7	..	1
Vagotomy.....	4	4	3	1	..
Previous Operation: Gastric Resection					
Vagotomy and gastric re-resection.....	3	2	2	..	..
Gastric re-resection.....	1	1	1	..	..

Of the four patients whose gastrojejunocolic fistulas followed gastric resection, three were treated by excision of the fistula with closure of the colon and jejunal openings, gastric re-resection and vagotomy. All had excellent results. One patient who underwent gastric re-resection also had an excellent result.

We were able to follow 15 of the 20 patients who had gastrojejunocolic fistula; 86.6% had an excellent result, 6.7% an unsatisfactory result and 6.7% a poor result. None had a recurrence of the fistula, and there were no operative deaths.

COMMENT

The present study indicates that vagotomy can be expected to give excellent results in 70% of cases of gastrojejunal ulcer after

adequate resection and carries a low operative risk. Whether vagotomy is done by the transthoracic or transabdominal route makes little difference as far as the ultimate result is concerned, but the objections to the transthoracic approach are that the surgeon is denied the opportunity of examining the gastrojejunal anastomosis to determine the true status of the ulceration or inflammation (for sometimes an ulcer is not present) and the exact amount of stomach remaining. Many of these patients have considerable postoperative thoracic pain which may last for many weeks and be most distressing.

The results of gastric resection are superior to those of vagotomy in cases of gastrojejunal ulcer after gastroenterostomy. Thus, in our series gastric resection yielded excellent results in 86.5% of 111 traced cases of this type as compared to 77.8% for vagotomy alone.

Our findings indicate that in more than 70% of the cases in which the gastrojejunal ulcer develops after gastric resection, vagotomy can be expected to give excellent results, with a low operative mortality rate (about 1%), whereas re-resection in this series carried an operative mortality rate of 15%. However, if re-resection is deemed advisable by the surgeon, then vagotomy may be added without greatly increasing the operative risk and may give the patient additional protection against further ulceration.

Significantly, in the vagotomy group free hydrochloric acid was present before vagotomy in all cases in which gastrojejunal ulcer developed after gastroenterostomy, and it was present in 77.9% of cases in which the ulcer developed after gastric resection. Vagotomy produced achlorhydria in only 28% of cases in which the ulcer had followed gastroenterostomy, and in 54.3% of the cases in which the ulcer had followed gastric resection.‖ It is likewise significant that in the resection group free hydrochloric acid was present before resection in about 95% of cases in which gastrojejunal ulcer developed after gastroenterostomy and in 75% of cases in which ulcer developed after gastric resection. Gastric resection produced achlorhydria in 80.2% ¶ of the cases in which the ulcers had followed gastroenterostomy. Gastric re-resection produced achlorhydria in 50% of the cases in which the ulcer had followed gastric resection. The greater incidence of achlorhydria undoubtedly is an important factor in the better results obtained after gastric resection than after vagotomy following gastroenterostomy.

Roentgenologic recognition of a gastrojejunal ulcer may be most difficult. The roentgenologist can detect a gastrojejunal ulcer after gastric resection more easily than after gastroenterostomy, but there is a high incidence of error in both. Therefore, a patient who has undergone gastroenterostomy or gastric resection for duodenal ulcer and who has recurrent symptoms should be suspected of having a gastrojejunal ulcer. If, in addition to these symptoms, he has had hemorrhage from the upper part of the gastrointestinal tract, a presumptive diagnosis of jejunal ulcer can be made and operation should be performed.

Gastrojejunal ulcer like duodenal ulcer tends to heal and to recur. Since operation is rarely necessary as an emergency procedure for gastrojejunal ulcer, the findings at the gastrojejunal anastomosis may not be pronounced, that is, there may not be an ulcer with a crater. However, in such a case the finding of inflammation in the region of the gastrojejunal anastomosis or of a scar indicating a healed ulcer should be sufficient evidence of gastrojejunal ulcer to justify treatment of such an ulcer by a definitive surgical procedure.

In the past the surgical treatment of gastrojejunocolic fistula has carried a high operative mortality whereas in this series of 20 cases there were no operative deaths. Of the 15 patients we were able to follow, 13 had

‖ This figure may be derived from Table 2 by subtracting the percentage of cases of achlorhydria before vagotomy from the corrseponding percentage after vagotomy.

¶ This figure may be arrived at from Table 2 by subtracting the percentage of cases of achlorhyria before resection from the corresponding percentage after gastric resection.

excellent results. This reflects not only improved surgical techniques, but demonstrates the important role of antibiotics, improved anesthesia, and better supportive measures that are available today.

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Surgical Experiences with Three Hundred Sixteen Duodenal and Marginal Gastric Ulcers

R. MORTON BOLMAN, M.S., M.D.
and
ROBERT P. LLOYD, M.D., Fort Wayne, Ind.

Hypersecretion of acid gastric juice has been proved by Dragstedt [1] to be a constant factor in patients with duodenal ulcer. The basic etiology underlying this constant is, unfortunately, not proved. Until the etiology is completely understood, as Walters [2] has pointed out, the modes of treatment and the results will necessarily vary.

The literature of the past 10 years is replete with excellent contributions, but the surgical field is still rather sharply divided into radical resectionists on the one side and the vagotomists on the other. Both sides are represented by excellent and conscientious men separated only by approach, not by fundamental concept.

Pavlov [3] and his group, in 1910, proved beyond question that acid gastric secretion occurs in animals approximately five minutes after the sight, smell, or taste of food, or even the appearance of the feeding attendant. He also proved that this modality was abolished if the vagus nerves were sectioned. This is the so-called psychic, or cephalic, phase of gastric secretion and is mediated through the vagus nerves. Beaumont noted this in his studies and a thorough investigation of the cephalic phase was made by Wolf and Wolff [4] in 1943. Hartzell [5] sectioned the vagi in dogs in 1929 and noted a marked diminution in free and total gastric acid.

As the food arrives in the stomach and makes contact with the pyloric antrum, the second, or gastric, phase of secretion is begun. It would seem that contact of the food with the antral mucosa is the essential trigger mechanism of the gastric phase. Dragstedt [6] and his co-workers in 1950 proved that exteriorization of the antrum causes a definite decrease in secretion, whereas transplantation of the antrum to the duodenum or colon causes rather marked hypersecretion. Ivy [7] felt that this averaged 225 to 350 cc. for a period of approximately five hours. This varies with the individual and, as Pavlov [8] has pointed out, with the type of food ingested.

The work of Edkins [9] in injecting an antrum extract intravenously to produce acid gastric secretion established the antrum as the initiator of the gastric phase and led to the postulation of a substance named "gastrin" as a humoral element responsible for the secretion. This was in 1906. In 1908, Edkins and Tweedy [9] divided the stomach into two separate compartments and found that food placed in the antral portion evoked the secretion of acid gastric juice by the fundic portion. The reverse is not true. This work has been repeated many times, most recently by Dragstedt [6] and his associates. They have shown that the antrum is a specific internal secreting organ, distinct in function from the mucous membrane of the remainder of the stomach and duodenum.

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Ivy and McIlvain [10] in 1923 proved the so-called intestinal phase of gastric secretion by the insertion of certain substances into intestinal fistulae.

Dragstedt [11] and his associates are convinced that the cephalic and gastric phases of gastric secretion account for 90% (equally divided) of gastric secretion. This leaves only an insignificant 10% to be accounted for by the intestinal phase. They believe that, in large part, the duodenal ulcer diathesis with its hypersecretion is the result of an overactive psychic or cephalic phase and can be abolished by complete interruption of the vagus nerves. This belief is based on the finding that resection of the antrum in the presence of duodenal ulcer is in many cases not followed by a substantial reduction of the hypersecretion so characteristic of the disease. Dragstedt [12] is convinced, however, that antrum resection plus vagotomy will uniformly reduce the secretion of the stomach remnant to a negligible amount. Dragstedt [11] is also convinced that the results of complete vagotomy are lasting. He presents experimental and clinical work to substantiate his belief.

Woodward and Bigelow,[14] working with Dragstedt, found that antrum resection produced a uniform decrease in secretions in Pavlov and Heidenham pouch dogs. This was found to be permanent.

Mann and Williamson,[15] in 1923, described a method by which jejunal ulceration could be produced in 90% to 100% of dogs operated on.

Storer,[16] working with Dragstedt, found that antrum resection alone protected about two-thirds of Mann-Williamson dogs from ulcers, while vagus section alone protected one-half. The combination of antrum resection with vagotomy protected all dogs in a series of Oliver's, personally reported to Dragstedt, and all but one in Storer's series. The combination of the two would then seem to be experimentally more efficacious in the prevention of ulcer than either alone.

Dragstedt [17] stresses the fact that after vagotomy the accompanying gastroenterostomy must be carefully and dependently placed, because retention stimulates the gastric phase, reinstitutes hypersecretion, and predisposes to marginal ulceration. Slight stoma dysfunction, in our hands, has not always been easy to prevent and seemingly bears little relationship to technique, experience, or standardization. One wonders if this phenomenon bears relationship to the *milieu interne* of Bernard or the property of homeostasis described by Cannon.

Ivy [18] says that 5% to 12% of our population may be expected to exhibit duodenal ulcers during their life span, and Mayo [19] stresses the magnitude of the problem by pointing out that, whereas 100 years ago most ulcers were gastric, now by far the vast majority are duodenal. Allen and Welch * believe that 15% to 20% of all patients admitted to the Massachusetts General Hospital with a diagnosis of duodenal ulcer will ultimately come to surgery.

Many surgeons have felt, and continue to feel, that high gastric resection is the treatment of choice for duodenal ulcer, but most, if not all, will admit to some dissatisfaction with their results. This dissatisfaction concerns itself chiefly with the difficulty of treatment in the event of recurrence, weight loss, the small stomach syndrome, the so-called "dumping syndrome," and a lack of feeling of wellbeing.

Crile,[22] of Cleveland, has reported a large series of vagotomies with gastroenterostomy with negligible mortality; recurrence less than with a similar group of high resections, and a high degree of patient satisfaction. Pollard,[23] of Ann Arbor, Mich., and his associates have reported similar results, as have Grimson [24] and his associates at Duke University.

Walters,[1] Ogilvie,[25] Allen and Welch,† and many others are staunch supporters of the high gastric resection.

Dragstedt,[26] in a recent publication, calls attention to the fact that in several cases of his series stasis due to gastroenterostomy

* References 20 and 21.

† References 20 and 21.

dysfunction was followed by a recurrence of hypersecretion, in spite of vagotomy. He states that this could have been avoided by adding resection of the antrum to the vagotomy.

Johnson and Orr,‡ of England, became dissatisfied with the incidence of the so-called small stomach syndrome and other undesirable side-effects of high gastrectomy and recently reported a sizable series of duodenal ulcers treated by vagotomy and conservative (50%) gastric resection with recurrence rate of 0.3% as compared to 1.0% in a similar group treated by high gastrectomy. Mortality was 1.5% in the conservative group and 4% in the radical group. Druckerman [29] and his group report a similar experience.

Practically all authors agree that when marginal ulceration follows gastrectomy, vagotomy is the treatment of choice.

PRESENTATION OF CASES

The cases we are presenting represent a personal series, all private patients, and with 94% personal contact follow-up study. This is possible because of the personal nature of our practice. We present these cases because there has been no mortality and because we believe our results compare favorably with those of high gastric resection, and this with an appreciably lower risk procedure. The results in our smaller series of conservative (50%) gastric resection with vagotomy have encouraged us to make this our "policy" operation for all duodenal ulcers seen during the past two years except in the presence of great age or severe concomitant disease. In this latter group, we still prefer gastroenterostomy and vagotomy.

Our series of cases consists of 341 operations on 316 patients with duodenal and marginal gastric ulcers. The only marginal gastric ulcers included in this series are those following our own original operations. The others will be reported elsewhere. This is a consecutive and unselected series, with the oldest patient 78 and the youngest 19 years of age. These cases divide themselves into three natural groups.

The first group of patients, 216 in number, had gastroenterostomy with vagotomy, all by the subdiaphragmatic abdominal route and all by the technique described by Dragstedt. These cases are broken down in Table 1. One hundred ninety of the gastroenterostomies were posterior and 26 were anterior. All were short loop operations, as nearly no loop as possible. One of us prefers isoperistaltic and the other an antiperistaltic anastomosis. We are convinced that whether the anastomosis is anterior or posterior, isoperistaltic or antiperistaltic makes not the slightest difference. Four of these patients, or about 20%, did not drain their stomachs for a protracted period of time, with the result that enteroenterostomy between the afferent and efferent loops was done. None of these four have had a recurrence; so it would seem that the bathing of the stoma with alkaline duodenal contents is most important when vagotomy is not done or vagotomy is technically incomplete. With more frequent and prolonged use of the Levin tube and the occasional administration of bethanechol (Urecholine), this irritating complication has been practically obviated. Seven, or 3.2%, have small bouts of diarrhea every three to six weeks. These are controlled by the periodic administration of succinylsulfa thiazole (Sulfasuxidine) and none are unable to work. Twelve, or 5.5%, have some degree of stasis varying from regurgitation of small amounts of bile to belching. None of these are disabled. Eleven, or 5%, developed marginal ulceration. Nine of these had immediate conservative gastric resection. Two had transthoracic vagotomy with the feeling that the first vagotomy had been incomplete. There was no improvement. Both were subsequently subjected to resection and are now well. There is no mortality in this group.

The second group, made up of 12 patients, had transthoracic vagotomy alone as definitive treatment for duodenal ulcer. These patients all had their primary operations in 1946. This group is represented in Table 2.

‡ References 27 and 28.

Seven of the 12 or 58%, had subsequent gastroenterostomy because of gastric stasis, vomiting and fullness. One, or 8.3%, developed a marginal ulcer with gastrojejunocolic fistula. Gastric resection, colon resection, and repair were done. The patient is now well. Two, or 16.6%, have mild symptoms of stasis not justifying further intervention. They are gainfully employed. There is no mortality in this group.

The third group had vagotomy with conservative (50%) gastric resection. There are 88 patients in this group. They are represented in Table 3. There have, as yet, been no recurrences. The longest period since surgery is eight years and the shortest is six months. Two, or 2.3%, have dumping syndromes that necessitate lying down after eating and the use of a high-protein, low-carbohydrate diet. Two, or 2.3%, have mild belching and regurgitation after meals and in the mornings. Neither justifies further intervention and neither is unable to work. There is no mortality in this group.

TABLE 1.—*Gastroenterostomy and Vagotomy*

Operation	Operations, No.	Excellent Results		Good Results		Recurrences		Mortality
		No.	Per Cent	No.	Per Cent	No.	Per Cent	
Gastroenterostomy and vagotomy	216	186	84.3	19	8.7	11	5	0
Enteroenterostomy (afferent to efferent loop)	4	4	2.0	..	...	..	..	0
Transthoracic vagotomy	2	0	0	0	0	2	1	0
Conservative gastric resection	11	11	5.0	0	0	0	0	0
Totals	233 operations on 216 patients *	201	91.3	19	8.7	Today, 0		0

* Since these statistics were compiled, a gastric resection was done on another patient in this group with a surgical mortality. The over-all mortality of this group is now 0.5%.

TABLE 2.—*Transthoracic Vagotomy*

Operation	Operations, No.	Excellent Results		Good Results		Recurrences	Mortality
		No.	Per Cent	No.	Per Cent		
Transthoracic vagotomy	12	3	25	2	16.6	0	0
Secondary gastroenterostomy	7	6	50	..	....	1	0
Gastric resection and repair of gastrojejunocolic fistula	1	1	8.3	..	....	..	0
Totals	20 operations on 12 patients	10	83.3	2	16.6	Today, 0	0

TABLE 3.—*Vagotomy with Limited (50%) Gastric Resection*

Operation	Operations, No.	Excellent Results		Good Results		Recurrences	Mortality
		No.	Per Cent	No.	Per Cent		
Vagotomy with limited (50%) gastric resection	88	84	95.4	4	4.6	0	0
Totals	88 operations on 88 patients *	84	95.4	4	4.6	0	0

* This series now numbers 104 cases. There has now been one death, for an over-all mortality of 0.9%.

COMMENT

We believe our results with gastroenterostomy and vagotomy are as good as could be hoped for with high gastric resection. There was no mortality. There is no patient unable to carry on a gainful occupation.

Since submitting this paper originally, we have had one death following gastric resection in a man vagotomized two years ago. He was operated on for recurrent bleeding. The

mortality in the vagotomy group now stands at 0.5%. Also since submitting this paper, we have had one death following conservative gastric resection with vagotomy. We have used this procedure in 104 such patients, so the over-all mortality for this group today is 0.9%.

Our series of high gastric resections done during the same time now numbers 112. There have been six deaths in this group, for an over-all mortality of 5.4%.

The fundamental thought that led to this presentation is that the primary operative mortality of high gastric resection in our hands is 4.5% to 5.0% higher than that of gastroenterostomy with vagotomy with results no better, insofar as recurrence is concerned, and not as good insofar as patient satisfaction is concerned. It would seem, then, that any attempt to improve the results of the surgical treatment of duodenal ulcer must be made, not in the direction of the removal of more and more stomach, but rather in the direction of an attempt to decrease mortality and undesirable side-effects by leaving more stomach behind. In addition to this, it seems logical to combine vagotomy with conservative gastric resection because the recurrences following either one alone are pretty well proved to depend on the remaining intact existence of the other. In this manner, one includes an operation to prevent recurrence with the operation for relief regardless of one's affinity for the school of vagotomy or that of radical resection.

On the basis of experimental work, previously cited, it would seem proved that recurrences following vagotomy and a drainage operation are the result of stimulation of the gastric phase mediated through the presence of the antrum. Conversely, it would seem that recurrences following gastric resection are the result of stimulation of the cephalic phase mediated by the intact vagus nerves. If this is true, interruption of the vagus nerves plus removal of the distal 50% of the stomach should eliminate marginal ulceration and recurrence, except in those cases where vagotomy is incomplete. In these few cases, transthoracic vagotomy is a simple, low-risk procedure to complete the abolition of the cephalic phase.

In our series of 216 gastroenterostomies with vagotomy as well as in our smaller series of 88 (now 104) conservative gastric resections with vagotomy, we have been impressed with the fact that these procedures can be done in an unselected group of people of all ages with a surgical mortality about one-fifth to one-tenth that of high gastric resection. The cure rate of the former procedure would seem to be as good as high gastric resection and that of the latter considerably better. When high gastric resection is done in the presence of perforation or hemorrhage, the risk is even higher. As a result, many average men hesitate to attempt definitive treatment in these situations, fearing the risk. If our results mean anything, the lower-risk operation might offer more opportunity for cure in the face of an emergency, with less operative risk.

In our hands, in a smaller series, we have been able to approximate the risk of gastroenterostomy and vagotomy by reducing the amount of stomach removed and combining a careful vagotomy. Severe side-effects of vagotomy have not been seen. Our patients with gastroenterostomy and vagotomy have no appreciable weight loss after six months. With conservative gastric resection and vagotomy, the average weight loss after six months is 4.8 lb. as compared with 13.8 lb. after high gastric resection.

Since a large portion of the acid-secreting cells of the stomach are in the fundus and since the trigger mechanism for the gastric phase lies solely in the antrum, it would seem that anything over 50% removal of the stomach does not rest on a firm physiological foundation.

When gastric resection has been done and marginal ulceration occurs, vagotomy is generally agreed to be the procedure of choice.

The undesirable side-effects and the mortality rate of gastric resection in our hands seem to bear a direct relationship to the amount of stomach removed. With these facts as a basis and our surgical experience

as clinical encouragement, the combination of conservative (50%) gastric resection combined with vagotomy seems to be a logical solution to the duodenal ulcer problem.

SUMMARY AND CONCLUSIONS

Three hundred sixteen cases of surgically treated duodenal ulcer are presented.

Two hundred sixteen had gastroenterostomy and vagotomy. In this group, there were 11 recurrences, all relieved by gastric resection. There was no mortality.

Twelve had transthoracic vagotomy in 1946. This is an unsatisfactory group, requiring 20 operations. There was no mortality.

Eighty-eight had conservative (50%) gastric resection and vagotomy. There have been no recurrences in this group. There has been no mortality.

Side-effects of vagotomy have been minimal.

On the basis of the proved anatomical and physiological facts concerning the mechanisms of secretion of gastric juice, it would seem that either high gastric resection alone or vagotomy and a drainage operation is inadequate surgical therapy. Since recurrences following resection depend on the presence of intact vagi and recurrences following vagotomy depend on the presence of the antrum, conservative (50%) gastric resection combined with vagotomy would seem to be the operation of choice in the treatment of the duodenal ulcer diathesis.

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DISCUSSION

On Papers by Drs. Walters, Chance, and Berkson and Bolman and Lloyd

Dr. Arkell Vaughn, Chicago: We performed our first vagotomy in May, 1946, and since then we have operated in 200 to 300 cases. In 1952 we reviewed our first 100 cases and tried to follow them up. Of the 100 cases we were able to follow up 97. In these 97 cases the age limits were 82 for the oldest and 18 for the youngest. We had 82 males and 18 females. We have been very cautious in selecting female patients because many of these frustrated women are the ones we have had trouble with in our vagotomies.

In the first 100 the approach was transthoracic in 5 and transabdominal in 95. The operation performed was vagotomy alone in 22 cases, vagotomy and posterior gastrostomy in 7, vagotomy and partial gastric resection in 6, and vagotomy and repair of gastrojejunocolic fistula in 2, and 2 cases had to be revagotomized. We are no longer doing only vagotomies but are doing more gastroenterostomies and vagotomies and more resections and vagotomies.

Concerning complications, it is said that the first 100 years are the hardest; so in the first 100 cases we had more complications. It was necessary to remove the spleen in 5 of our first 100 cases because of technical difficulties. We had to blame somebody, so we blamed the intern or resident.

Wound disruptions, six: in these six we had one death. We had two wound eviscerations. We had two cases of cardiospasm which responded to tetraethylammonium (Etamon). We had one hiatus hernia which had to be reoperated on. A vicious circle occurred in five cases. These, we think, were due to technical difficulties. We let these cases alone for 21 days and after that time there was emptying. We had to revagotomize for previously incomplete vagotomy in two cases.

A dumping syndrome followed gastric resection in two cases. Cholecystectomy for persistent pain due to cholelithiasis was performed in two cases. If a patient has had a complete vagotomy and has further abdominal symptoms, those not favoring the procedure blame the vagotomy. Many times the trouble is due to some other cause, such as cholecystitis.

Diarrhea has not been a severe complication in our series. We had three or four with this complication. We find that diarrhea following vagotomy is controlled best by dilute hydrochloric acid taken in water during meals. Distention after retention of food was controlled by keeping the tube in for five days and giving methacholine (Urecholine).

I want to stress the importance of running the Hollander insulin test before performing a vagotomy. Many surgeons will say they did a vagotomy and didn't get good results. You will ask them if a complete vagotomy was performed. The crux of the operation is to do a complete vagotomy. They don't know if a complete vagotomy was performed.

"Did you run any Hollander insulin tests?"

"No, we didn't."

We think the Hollander test should be run before and after the operation to determine whether or not we have succeeded in getting all the vagus fibers.

Of the first 33 cases, we ran the night secretion and the Hollander insulin test. We found the volume of night secretion before vagotomy was 576.7 cc., and after vagotomy it was 336.7 cc., showing that vagotomy does cut down gastric secretion. The free acidity averaged 53.6% before vagotomy and 3.37 after vagotomy, but 30 of those cases had no free acid and 3 did, indicating that 3 of these cases had an incomplete vagotomy.

Dragstedt states that a complete division of the vagus nerve supply reduces the output of hydrochloric acid 96%. It sometimes is difficult to get all fibers, and some surgeons miss them; in fact, Dr. Ed Miller and Dr. Carl Davis Jr. several years ago reported the anomalous distribution of the vagus nerve. In spite of that, many times the fibers are missed; so if the insulin test is run afterward we have an insight as to how successful we were in getting the vagus fibers.

We have been doing the new procedure of Dragstedt, which is making an incision in the diaphragm above the hiatus into the posterior mediastinum, putting the finger in, freeing the esophagus, and getting the fibers, hoping that by this procedure

we will get higher up on the trunk before the branches occur.

Our results in these 97 cases: Seventy-seven of them were excellent. We consider good results to be what Dr. Bolman said, namely, these patients who say, "I feel good, and I don't have much trouble. I am working, and I think it is a good operation." We have a total of 88 that had good to excellent results. We have nine poor results, and one of the patients died from evisceration and one from an obstruction which might have been a mesenteric thrombosis. We didn't have enough nerve to try to remove the mesenteric thrombosis as was reported yesterday. Complications occurred mainly in frustrated women. The poor results occurred mainly in women. For that reason we are rather hesitant about operating on them.

In closing, may I stress the importance of getting all the vagus fibers and doing a complete vagotomy and running the Hollander insulin test, which will determine how much, if any, acid is left.

Dr. John Reynolds, Chicago: I asked Dr. Bolman for the privilege of discussing his paper for three reasons. First, I think he has reported to us a commendably low death rate. I believe a zero mortality should be our constant aim in all elective operations for ulcer. Secondly, he has emphasized that these patients should have no more ulcer pain. And, finally, the operative and nonoperative care of these patients should envision an end-result in which the patients are able to eat well, are able to work, and have a minimum of postgastrectomy disturbance.

One of the things which strikes me as being very important is that surgeons must realize that each individual patient probably has an individual problem of hypersecretion. Thus, one patient may produce 1500 to 2000 cc. of gastric secretion in a 12-hour period and another patient no more than 600 cc. There are probably other individual factors which are difficult to measure, such as the amount of gastric secretion produced by individual patients, and there are also probably variations in the ability of the patient's duodenum to resist qualitative and quantitative excesses of gastric secretion. Since this is true, one can expect different responses in different patients to a standard operation.

With such a basic tenet, it must be realized that no operation can be depended upon to give 100% satisfactory results to every patient and that every patient must realize that to a certain extent some cooperation in the line of dietary precautions may be necessary. Poor care of a patient postoperatively from a medical point of view may give as bad a result as a poor surgical operation, and a combination of these two, of course, can be depended upon to give a poor result. We have had two patients who are illustrative. One was a very highstrung man, who had obtained an excellent result both from the Hollander test and from the symptom analysis after an operation which included a vagotomy and a gastroenterostomy. On his honeymoon, he went to France and drank a great deal of champagne and smoked 30 cigarettes a day. His recurrent ulcer disappeared at once upon his return, when he was requested to be a little more reasonable in his diet. Since he has recognized that he cannot violate every law of gastrointestinal physiology, he has remained well. The second patient developed recurrent symptoms after gastric resection and a vagotomy. It was discovered that his daily regimen was to get up in the morning, smoke five cigarettes before breakfast, then have a breakfast consisting of a large glass of orange juice, two bowls of very strong chile and several cups of black coffee. When it was pointed out to him the necessity of neutralizing acid with a bland protein early morning feeding, he immediately lost all of his ulcer symptoms and began to gain weight for the first time.

Last, but not least, Dr. Bolman should be particularly complimented about his work. He does not have a big university with a professor pressing him. He does not have a big clinic to urge him on. He does not have a Veterans Hospital.

He is working with his own patients in his own community and I think he has done an excellent job.

Dr. Chester C. Guy, Chicago: It sounds as if the Chicago contingent has some pretty definite ideas about this subject. I am very happy to have had the privilege of hearing these two very excellent papers. I noted with interest in Dr. Chance's paper that the essayists stated that the average time interval between the operation of gastric resection and the development of gastrojejunal ulcer as far as they could tell was some three years, whereas the average time between a gastroenterostomy and the development of a gastrojejunal ulcer was in excess of 11 years, or over three times as long. This of course suggests what we already know—that in the evaluation of our results it is the long-term follow-up that is going to give the final answer to this controversial question as to which is the preferable operation for the treatment of duodenal ulcer.

I wish to congratulate and compliment Dr. Bolman on the excellent results he has had and the fine presentation which he made. In some 316 patients, I believe, subjected to vagotomy and gastroenterostomy or resection he had only 2 deaths. I hope his success in the future will continue to be as good, but I doubt if it will. [Laughter]

Our experience with very limited resection and vagotomy does not warrant any conclusions, but I should like to comment on our results with subdiaphragmatic vagotomy and gastroenterostomy for duodenal ulcer.

[Slide] We started this operation about seven years ago, and about two years ago we compiled our results by what we believe to be a reasonably and accurate and careful follow-up of 168 patients. We were agreeably surprised to find that 91% of them were in satisfactory condition, either excellent or good. We had not anticipated it would be that high.

It also was of interest to us to note that the percentage of satisfactory results was about the same, regardless of the duration of time since the operation.

[Slide] Last winter we again surveyed our experience with this operation for duodenal ulcer and were able to follow 253 patients with reasonable accuracy for a period of from six months to six years. The results were the same, 91% satisfactory. These are almost the identical results reported by Dr. Bolman and those which have been reported by others that have been mentioned today.

These results were as good as we had ever been able to obtain from a two-thirds or three-fourths gastric resection, and, in addition, we believe that the nutritional status of these patients months or years after their operation was superior to that of the patients who had resection.

On the basis of our experience with gastric surgery, we have come to the conclusion that subdiaphragmatic vagotomy and gastroenterostomy (at least as of today) is the treatment of choice for the great majority of duodenal ulcers.

DR. KENNETH JENNINGS, Santa Barbara, Calif.: I come from the Far West, where in order to survive one has to learn to see clearly through the fog. [Laughter]

I wish to call attention to the point that in evaluating ulcer surgery it is unfair to include recent results. In my opinion, one should not include results that are not at least two years beyond the date of surgery.

Only last week we operated on a patient on whom we had done a gastroenterostomy and a vagotomy some three years ago. At the termination of the operation a Hollander insulin test was done, and the patient was practically antacid. As I recall he had 3 units of free hydrochloric acid. One year later he was still well and would have been classed as certainly a good, if not an excellent, result.

In the past year, however, he has had two severe hemorrhages, and at operation last week we found a well-developed jejunal ulcer. So, I feel it is unfair in evaluating these results to include the 30 or 40 cases which obviously had been added since the first writing of the manuscript.

DR. G. ARNOLD STEVENS, Beverly Hills, Calif.: I am interested in the excellent paper of Drs. Bolman and Lloyd and particularly that their experience suggests that the best results in the surgical treatment of duodenal ulcer follow conservative gastric resection combined with vagotomy. This parallels our own experience.

In a report of a small control series of hemigastrectomy of the Polya type with subtotal vagotomy of both vagus nerves published in 1948 and in subsequent reports, we submitted that the more radical adequate gastric resection should not be discarded generally. In the light of subsequent experience we wish to retract this statement. As of last week we had studied 107 cases of hemigastrectomy with subtotal vagotomy with an average six-year postoperative follow-up. The operations were performed at St. John's Hospital and at the Los Angeles Veterans Hospital (Wadsworth). There has been only one marginal ulcer in this series. This patient has had no recurrence following a supplemental transthoracic so-called total vagotomy six years ago. He has had motility disturbances.

On this basis it would appear that had we attempted total vagotomy on all of these cases there would have been no incidence of marginal ulcer so far in this series. How this would have affected the good results so far as motility is concerned is problematical. I should like to have the opinion of Drs. Bolman and Lloyd regarding this.

Postoperative data have included barium x-ray studies. There has been no instance of gastric retention.

My associates, Drs. John Briggs, Philip Beale, and Newlin Hastings, have more recently supplemented this study with a series of 42 cases treated by Bilroth I hemigastrectomy and as complete a vagotomy as possible. This phase of the study is being continued. The morbidity so far in this group has been more than it has been with the Polya type of procedure in our larger series. In 4 of 42 cases it has been necessary to replace the gastroduodenostomy with a gastrojejunostomy to correct inadequate emptying of the stomach.

Since we now know that occasional marginal ulcers occur even after the more radical so-called adequate gastric resection with supplemental total vagotomy, it is evident that nothing short of total gastrectomy will completely prevent this.

From a practical standpoint, however, it does seem at the moment that conservative gastrectomy combined with some form of vagotomy constitutes a procedure with the minimum anatomic and physiologic mutilation which is consistent with optimum results.

DR. R. MORTON BOLMAN, Fort Wayne, Ind.: I should like to thank the discussants very much for their remarks. I would like to say that I agree 100% with Dr. Jennings. The only reason that the cases were added after the manuscript was written was to show operative mortality and nothing whatsoever so far as cure of the disease is concerned.

That is why I mentioned that I think only 47 of these gastric resections are of any significance whatever, because there are only 47 of them that are longer than two and a half years.

Dr. Chance mentioned something about vagotomy, either transthoracic or subdiaphragmatic, for marginal ulcer following gastroenterostomy. I should like to say that we had one disastrous experience doing a transthoracic vagotomy for recurrence in an elderly woman following gastroenterostomy without vagotomy. She had her gastroenterostomy done elsewhere and had developed a marginal ulcer. She was in rather a delicate state of health, and we elected to do a transthoracic vagotomy for treatment of her ulcer.

It worked out later that she had a carcinoma at the margin of her stomach, and by the time we realized it it was too late to apply any efficacious treatment. We lost a patient that we might otherwise not have lost.

In the problem of recurrence in the poor-risk patient, I wish to mention in passing that we have had five such patients, three of whom have had major coronary episodes within five years of the time of difficulty. One of them had had his gastroenterostomy 27 years previously, and the new stoma was being occluded with an ulcer. The duodenum was occluded with the scar tissue of the original ulcer.

Three had coronary episodes. One had coronary insufficiency, and one had one kidney with a stone, with blood chemistry values that were very discouraging.

In these five patients we did subdiaphragmatic vagotomy, took down their posterior gastroenterostomies, merely cutting across the jejunum on each side of the ulcer, turning the ends in and leaving the ulcer alone, reanastomosing the jejunum end-to-end, and doing an anterior gastroenterostomy with the vagotomy.

Whether that is a good procedure or not, I don't know, but the five persons are alive, and I believe the youngest is 69, the oldest 74. We were able to get by with those five patients without mortality.

Subtotal Gastric Resection with Colon Substitution

Reconstruction of a Neostomach Utilizing the Transverse Colon

DAVID H. WATKINS, M.D.
and
GEORGE WITTENSTEIN, M.D., Denver

Until recently surgeons all over the world generally agreed that an extensive gastric resection (75 to 80%) was the procedure of choice for peptic ulcer, the operation usually being carried out on the Billroth II plan. The only other operation that has had any general acceptance, and this has been principally in Germany and Scandinavia, is the Billroth I gastric resection. It is agreed that the extensive subtotal gastrectomy renders the patients free from their previous symptoms and greatly decreases the chances of recurrent jejunal ulcer. However, a majority of such patients do not gain weight and they have minor postcibal symptoms. The so-called postgastrectomy syndrome includes not only certain postcibal symptoms but also a considerable loss of the ability to gain weight and renders these patients incapable of work demanding great activity. These patients have been regarded as suffering from a gross disorder of physiology due to the exclusion of the duodenum (Hudson).[1]

Zollinger [2] emphasized that many gastric operations produced distressing postoperative complications unrelated to the original disease. A direct relationship between type of surgery and nutritional status was observed. He found that the best weight gains occurred in patients having vagotomies and gastroenterostomies or in those with vagotomies and 60% gastric resections and gastroduodenostomies.

Particularly since the end of World War II, numerous reports have been documenting the occurence or exacerbation of pulmonary tuberculosis following subtotal gastrectomy (Gordet).[3] Postgastrectomy marasmus has been reported by Moroney.[4]

It must be emphasized that not all postprandial symptoms complained of by patients who have undergone subtotal gastrectomies are sequelae of the operation or recurrences of their peptic ulcers.

Formerly the emphasis was centered on employing an operative method which secured the patient the greatest possible protection against recurrent ulceration, while other late postoperative disturbances, such as anemia, postcibal distress, and nutritional disturbances were accorded less importance and often regarded as the price to be paid to obtain cure of the peptic ulcer. In more recent years, however, these postgastrectomy complications have called for greater interest, particularly since the number of patients suffering from these disorders has increased in proportion to the increasing number of gastric resections performed. At the present time more and more attention is being given to these complications, and means for preventing their occurrence are of increasing importance.

From the Department of Surgery, University of Colorado Medical Center.

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The symptoms which occur after eating in patients who have undergone subtotal gastrectomy may be divided into those which occur early after eating and those which occur at a later time following the ingestion of food. Meurling [5] found that postcibal symptoms occur more than 45 minutes to 1 hour after eating. The causes of these symptoms may be classified as follows: (1) Symptoms which originate principally from the small intestine. Among these symptoms are included the following syndromes: dumping stomach, jejunal distention, irritation of the small intestine by increased motor activity, splanchnic hyperemia, jejunitis, allergy, afferent loop reflux, afferent loop stasis. (2) Symptoms arising mainly from gastric remnant: gastric distention, gastritis, achylia, stretching of the gastric remnant. (3) Other causes: hypoglycemia, hyperglycemia, anemia, neurologic and psychic factors, and miscellaneous factors such as decreased serum potassium. After a critical study of the literature, he concluded that many of these factors may contribute to the occurrence of postcibal symptoms but that none of them can be regarded as having proved decisive significance in the origin of these symptoms.

The occurrence of such postcibal symptoms following partial or subtotal gastrectomy and their incidence in different series varies according to a definition of the syndromes or the corresponding nomenclature used by those reporting them, as well as upon the thoroughness with which the patients are questioned concerning symptoms. In the literature the reported incidence varies from 5 to 75%. Meurling found 32.7% had no postcibal symptoms; 26% had slight or latent symptoms, and 41.3% had definite postcibal symptoms. His observations were based on a follow-up of 624 cases, including both Billroth I and Billroth II types of gastrectomies. His figures agree with those of earlier investigators (Friedemann,[6] Hoffman,[7] Watson [8] and Muir [9]).

Recently Wallensten [10] has attempted to evaluate whether the results of the Billroth I and Billroth II types of operation for the relief of peptic ulcer are equally good or if one is to be preferred in certain cases. In his series of 203 Billroth I partial gastrectomies there were 17 cases that could be classified as recurrences (8.4%). In the Billroth II series of 322 cases there were 8 recurrent cases (2.5%). The recurrence incidence following Billroth I partial gastrectomy proved to be higher than after Billroth II. This applied especially to duodenal ulcer in men. In gastric ulcer in men and in both types in women the recurrence incidence was found to be probably the same whether a Billroth I or a Billroth II subtotal gastrectomy is done. Postoperative anemia occurred more frequently after Billroth II than Billroth I partial gastrectomy; this difference was particularly pronounced in women. It was demonstrated by the investigation of Meurling [5] that the postoperative anemia may conceivably be due to defects of postoperative iron absorption. Postcibal symptoms occurred more pronouncedly and in severer grades following Billroth II than following Billroth I partial gastrectomy. There is reason to believe that an iron deficiency may be one of the factors contributing to the origin of the postcibal symptoms. Changes in weight as manifested by difficulty in maintaining preoperative weight occurred much more frequently following Billroth II than Billroth I partial gastrectomy. This investigation also demonstrated that there is a difference in the results of a Billroth I and a Billroth II partial gastrectomy, even if the extent of the resection is the same with the two methods. On the basis of the results of this investigation, Meurling recommended that better results would be obtained from resection therapy of peptic ulcer if a Billroth II partial gastrectomy were done for duodenal ulcer in men and a Billroth I partial gastrectomy were performed for gastric ulcer in men and both types of ulcer in women.

Correspondingly, attempts have been made to devise a procedure which would combine the virtues of subtotal gastrectomy and yet propel food into the duodenum. To obtain a recurrence rate as low as 4%, 75% to 80%

of the stomach must be removed by subtotal gastrectomy, followed by some type of Billroth II anastomosis. The duodenum and 12 in. of jejunum are excluded from the food stream. The crop function of the stomach is destroyed and no, or only a partial, stomach reservoir is left. Food is not macerated and passed gradually to the duodenum but rapidly slips into the small intestine in essentially the same form in which it has been swallowed. The deficiencies in basic food substances are thus due to inadequate intake, inadequate digestion, and failure of absorption; the rapid passage into the jejunum contributes to further rapid passage throughout the small intestine.

Among the procedures which have been advocated to replace the routine gastric resection are gastroenterostomy, vagotomy alone or with a drainage procedure such as pyloroplasty or gastroenterostomy, hemigastrectomy with vagotomy, and conventional subtotal gastrectomy with vagotomy.

More recently modifications of the classic subtotal gastrectomy have been suggested as alternatives. Connell [11] suggested "fundusectomy" to excise the acid-secreting area of the stomach. Wangensteen [12] suggested a modification of Connell's procedure in which the entire greater curvature from the area immediately to the left of the esophagus to the antrum is excised, converting this segment into a tube. Recently, however, Wangensteen [13] demonstrated that Connell's fundusectomy and his previous tubular resection did not remove enough of the acid-secreting area to protect against recurrent ulcer. He presented a revival and modification of the segmental gastric resection of Mikulicz [14] in which not more than 10% of the entire stomach or 15% of the acid-secreting area is preserved. This procedure was accompanied by an adequate pyloroplasty, since the excision of a wide gastric segment deprives the pylorus and antrogastric fragment of its vagus supply. In summing up the evaluation it was felt that the percentage of excellent or satisfactory results was almost identical with those obtained with the Billroth II operation.

Recently, several attempts have been made to treat the complicated duodenal ulcer by an extensive gastric resection followed by a procedure which attempts to restore the crop function of the stomach and reestablish normal gastrointestinal continuity. In 1951 Moroney [4] reported the use of the transverse colon to replace and restore the excised stomach and to establish the alimentary canal, providing a continuous channel between the gastric remnant and the duodenum. The use of a segment of colon restores the reservoir or crop function of the stomach; it preserves function of the duodenum and minimizes interference with small bowel function. The resistance of the colonic mucous membrane to acid peptic digestion has been demonstrated by cases in man of gastrojejunocolic fistula in which the repair of that fistula resulted in segments of colonic mucosa being implanted in the stomach wall. Some of these cases have been followed for seven years and gastroscopically and radiographically show no abnormality.

Henley [15] in 1952, in an attempt to devise a procedure which would retain the virtues of subtotal gastrectomy and yet enable food to enter the duodenum as in a Billroth I type of operation, used segments of jejunum varying between 4 and 8 in. in length, according to the amount of stomach and duodenum removed.

Stimulated by our [16] experiences in using the transverse and left colon for restoration of gastrointestinal continuity in cases of total gastrectomy, we proposed a trial of transverse colon replacement for the excised distal segment following subtotal gastrectomy.

OPERATIVE TECHNIQUE

Resection of the stomach is carried out according to the indications of the presenting pathology. In the case of duodenal ulcer, a 75% gastrectomy is performed, removing both vascular arcades and the lesser curvature of the stomach, so that a gastric stoma is formed whose diameter approximates that of the transversely sectioned colon. In the case of carcinoma of the stomach, involving

Fig. 1.—After distal subtotal gastrectomy the duodenum has been transected in its first portion and the proximal gastric remnant remains. In the classic reconstruction the duodenal stump has been closed and a gastrojejunostomy has been made to reestablish gastrointestinal continuity (Billroth II type of reconstruction).

the pyloric third, a 75% distal subtotal gastrectomy is carried out, with removal of the greater omentum, detaching it from the transverse colon by the Hey Groves maneuver. In cases of carcinoma of the stomach involving the middle and proximal thirds, total gastrectomy is performed by the combined thoracoabdominal approach and intestinal continuity is reestablished by utilizing a portion of colon as previously described by us. In those cases of carcinoma of the stomach which secondarily have invaded the colon, the involved portion of the colon, together with its mesentery, is resected en bloc. Such a maneuver, however, does not exclude the use of another portion of the colon as a gastric replacement.

The colon secretes a considerable amount of mucus, which is particularly potent in neutralizing the secretions and enzymes of the upper gastrointestinal tract. It has the advantage of being markedly distensible, and our experience has demonstrated that it can be used in an antiperistaltic direction. The result of the interposition of a segment of colon between the remaining gastric remnant, or, in the case of a total gastrectomy, between the esophagus and the duodenum, is that bile and the alkaline secretions of the upper small intestine are prevented from retrograde inundation of the lower esophagus to produce an alkaline esophagitis (Lortat-Jacob).[17] This plan of utilizing the transverse colon would appear to be more circumspect than the use of a free loop of jejunum because of the susceptibility of jejunal mucosa to peptic digestion.

After mobilization of the stomach, the duodenum is transected and the entire viscus turned to the left (Fig.1). Before the proximal end of the stomach is transected, a suitable portion of the transverse colon is selected with regard to adequate vascularization. A small hemostat is passed through an avascular portion of the mesocolon close to the bowel. Two Allen clamps are placed obliquely across the transverse colon at its right extremity and a wedge of transverse colon on the antimesenteric border excised. A similar procedure is carried out at the left extremity of the selected segment of transverse colon. It has been unnecessary to divide any major colic vessels, and no mesocolic incisions are necessary. After the division and isolation of the colon segment, the butt ends of the remaining colon are approximated end-to-end fashion in one or two layers. The isolated segment of the transverse colon is then rotated 180 degrees so that it comes to lie nicely in the supramesocolic compartment of the abdomen.

The coloduodenal anastomosis in most cases is a very simple matter. It may be done in one or two layers. If the duodenum is too large after transection, the well-known method of slightly wider bites on the larger side obviates it. If the ulcer is adherent posteriorly, and this is frequently the case, the duodenum is transected in open fashion without the use of clamps. No attempt is made to remove the ulcer crater. If the duodenum can be mobilized so that an adequate anterior wall can be obtained, anastomosis or closure can be accomplished. If the duodenum is small and narrow, it is cut along its lower border; otherwise, stricture and obstruction can supervene. The coloduodenal anastomosis can be accomplished in one or two layers. The most important consideration is that this anastomosis be performed with interrupted sutures throughout. It is perfectly safe to suture the colon to the crater of

the ulcer rather than to attempt a difficult mobilization from the head of the pancreas. In many cases, if a single layer of sutures is desired, a vertical mattress suture in which the first stitch includes all layers of the walls of the two viscera and the second includes only the serosal or muscular layers, the result will be effective invagination of the mucosa. In difficult cases in which the duodenum is adherent posteriorly, the anastomosis may be accomplished by horizontal mattress sutures.

After the completion of the coloduodenal anastomosis, the portion of the stomach to be resected is determined. An Allen clamp is placed at right angles to the greater curvature and marks off a gastric stoma equal in length to the divided transverse colon. From the end of the Allen clamp a Furniss clamp is placed obliquely between that point and the esophagus so that the entire lesser curvature is removed. The lesser curvature is sutured in two layers. The gastrocolic anastomosis is usually accomplished in two layers. Upon completion, all anastomoses are buttressed by the apposition of tabs of omentum (Fig. 2). All that remains to be done is to make sure that no rent exists in the transverse mesocolon. A 1-in. cigarette drain is placed in Morrison's pouch and brought out through a stab wound in the right flank. Care should be taken that this drain does not abut upon the coloduodenal anastomosis.

Fig. 2.—In the method of reconstruction proposed here the same type of resection is carried out as in Figure 1, but continuity is reestablished by intercalating a segment of transverse colon between the gastric stoma (fashioned in the Hofmeister-Finsterer manner) and the duodenal orifice. The segment is rotated 180 degrees after sectioning for ease in anastomosis.

POSTOPERATIVE CARE

No special considerations are necessary except that the patient should be ambulated the evening of the day of surgery and the diet should begin with semisolid food, liquids being withheld until after the completion of the solid portion of the meal. Alimentation may begin as soon as flatus is passed by rectum. Many patients have symptoms of gastric delay or atony, which diminish gradually over the first three postoperative months. These symptoms usually appear as complaints of epigastric fullness and discomfort in the left upper quadrant, which improves spontaneously after half an hour to an hour after the ingestion of the meal. Many patients find that they are unable to take cold liquids without precipitating rather severe epigastric cramps. In most patients we have found that the immediate postoperative status is improved by the exhibition of belladonna and other antispasmodics.

The gastric capacity is a composite of the capacity of the gastric remnant plus that of the colon loop. Retrograde moments occur, and their effects appear to be beneficial. The pattern of small intestinal motility appears normal. The secretion from the stomach shows an excess of mucus and achlorhydria in most patients.

CASE 1.—I. M. C., a 64-year-old arthritic white woman, entered Denver General Hospital on Oct. 26, 1953, with the complaint of cramping lower abdominal pain of 12 hours' duration. There was no stool or flatus since the onset of the pain. Two weeks before admission she passed tarry stools, followed by grayish stools. No food intolerance was elicited, and there was no history of alcohol or epigastric pain.

Examination showed an acutely ill woman with the characteristic hand and wrist deformity of rheumatoid arthritis. Her respirations were shallow and grunting. Her temperature was 37 C (98.6 F). The pulse was 70; blood pressure was 175/65; respirations were 22. The tongue was slightly dry, but there was no severe dehydration. There was tachycardia, with gallop rhythm and a Grade 2 systolic murmur to the left of the left sternal border in the fifth interspace. The murmur was not transmitted. The abdomen was board-like and without bowel sounds. Diffuse rebound and direct tenderness were universally present.

Fig. 3 (Patient I. M. C.).—Roentgenogram Dec. 10, 1953, shows some dilation of the gastric remnant and a spastic, contracted segment of colon between the gastric remnant and the duodenum.

A diagnosis of perforated peptic ulcer was made, and suture of a perforated prepyloric ulcer was carried out. The postoperative course was uneventful.

On Nov. 24, 1953, a subtotal gastrectomy with gastrocoloduodenostomy utilizing a 20-cm. segment of transverse colon was performed.

Pathological examination of the resected stomach demonstrated a hemorrhagic chronic peptic ulcer in the prepyloric area.

An upper gastrointestinal series on Dec. 10, 1953, showed that the stomach pouch was somewhat dilated and the anastomosis between the fundus of the stomach and the colon pouch was small but presented no evidence of obstruction. The duodenum was entirely normal (Fig. 3). The emptying time of the entire neostomach was normal. Postoperatively the patient complained of stomach cramps and regurgitation, which were controlled with phenobarbital and belladonna.

Gastroscopy on Aug. 6, 1954, showed the remaining gastric mucosa to be quite normal with no ulceration, bleeding, or gastritis. The colon anastomosis was easily visualized, and the mucosa of the colon was flattened and appeared to be normal with no evidence of hemorrhage, ulceration, or bleeding.

An upper gastrointestinal series on Aug. 16, 1954, showed the remaining stomach pouch and the transplanted portion of the colon functioning very well (Fig. 4). There was good churning action in the colon segment. No evidence of obstruction at the anastomosis was seen.

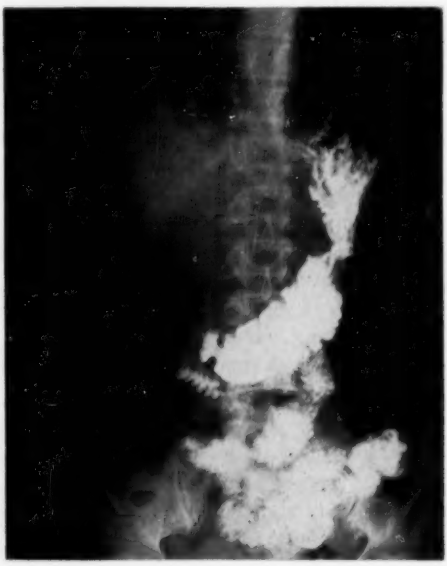
Her weight had increased from a preoperative level of 105 lb. to 115 lb. No free acid was present in the gastric aspirate. The provocative pepsinogen test showed a resting level of 60 tyrosine units. Stimulation produced no increases over a period of four hours.

Comment.—This patient developed the usual postprandial cramps which we believe to be characteristic of any colon transplant, whether it is anastomosed isoperistaltically or antiperistaltically. This distress disappeared after a period of three months, and she has remained free of dyspeptic complaints.

The change in appearance of the gastric remnant between December and August is interesting. The intercalated segment of colon had become less spastic and the gastric pouch had decreased considerably in size.

Case 2.—L. H., a 43-year-old white male alcoholic, spotter in dry cleaning establishments, had had a diagnosis of duodenal ulcer since 1930; a duodenal perforation was repaired in 1938. He was free of symptoms from 1938 to 1940, but since that time he had had a chronic ulcer history. He had large hematemeses in 1944 and 1946 and a minor

Fig. 4 (Patient I. M. C.).—Roentgenogram of Aug. 16, 1954, shows loss of spasticity of the colon implant and a decrease in size of the gastric remnant.



hematemesis five days prior to admission. After an alcoholic debauch of 20 days' duration he developed pneumonia and was admitted on Dec. 28, 1953. He had had chills, fever, hemoptysis, and severe cough. He had had frequent dark and tarry stools after drinking bouts. He usually weighed about 140 lb., and his appetite was always good.

Examination disclosed a florid, anxious, dyspneic, middle-aged man in acute respiratory distress. He coughed frequently and expectorated rusty sputum. His temperature was 39.4 C (102.9 F); blood pressure was 190/60; pulse was 112; respirations were 40. Decreased expansion of the right chest with splinting of the right diaphragm was noted. Bronchial breath sounds, dullness, whispered pectoriloquy were present on the right. The abdomen was negative except for a right paramedian scar.

After subsidence of the pneumonia the patient complained of marked left upper quadrant pain beneath the rib margins, severest in the evening. He complained of a burning sensation in the epigastrium, coming on 30 minutes to 1 hour after food, without radiation. He was only partially relieved by alkalies.

Urinalysis showed an occasional leucocyte. The hemoglobin was 14 gm. per 100 cc. Total leucocytes were 13,500, with 64% polymorphonuclears. The total protein was 5.4 gm. per 100 cc. with 4 Gm. albumin.

A gastrointestinal series showed very coarse mucosal folds throughout the stomach and some shortening of the lesser curvature. The duodenal bulb was definitely deformed, but no evidence of an active ulcer crater was seen. Follow-up films at one and two hours revealed normal progress of the barium meal in the small bowel. No significant retention in the stomach was seen.

Because of the long history of ulcer which had not responded to medical management and because of its recent exacerbation, which again failed to be symptomatically improved on medical therapy, operation was advised and was carried out on Jan. 26, 1954.

Numerous dense adhesions were present in the upper abdomen, especially involving the pylorus and duodenum; these were freed up and the stomach and duodenal wall exposed. There was a duodenal scar in the anterosuperior portion of the first part of the duodenum. The omentum was separated from the greater curvature in the usual manner, with ligation of the right and left gastroepiploic vessels. The left gastric vessels were similarly ligated and divided, as was also the gastrohepatic omentum. Next, the gastrocolic omentum was separated from the transverse colon, and a 15-cm. length of colon was prepared. This segment of colon was sectioned between clamps, after ligation of appropriate vessels and mesocolon. The stomach was transected in the usual manner, by applying an Allen clamp to the duodenum and dividing the proximal gastric segment according to the Hofmeister-Finsterer procedure. The lesser curvature of the stomach was reconstructed in three layers. The segment of transverse colon was intercalated between the proximal gastric pouch and the duodenal stump, using two layers of sutures. The colon was reconstructed by an end-to-end suture in two layers. A Levin tube was passed in the colonic pouch. After the placement of retention sutures the abdomen was closed.

The postoperative course was uneventful. However, the patient developed postprandial cramps, especially after the ingestion of cold liquids. He regained his preoperative weight of 145 lb. within six weeks and maintained it.

Upon examination on April 14, 1954, no free acid was present in the gastric aspirate. The provocative pepsinogen test showed a resting level of 55 tyrosine units. Stimulation produced no increases over a period of four hours. An upper gastrointestinal series showed a normal remaining stomach pouch and the transplanted portion of the colon functioning very well. Churning action was vigorous in the colon segment. The neostomach did not empty completely until six hours.

CASE 3.—A. P., a 64-year-old retired white male cement finisher, was admitted to the hospital with the complaint of abdominal pain of four hours' duration on Jan. 20, 1954. He had been previously treated in May, 1953, at the Denver General Hospital for a bleeding peptic ulcer, and he was placed at that time on a medical ulcer regimen. He had no vomiting or diarrhea, but his stools were positive for blood. The pain was constant, and there was some radiation into his back.

Physical examination demonstrated a well developed, well nourished white man, who was doubled over with severe pain and who answered questions with gasping speech. His blood pressure was 140/90; pulse was 88; respirations were 26 per minute. The chest was clear. Heart sounds were normal. The abdomen was scaphoid, rigid, and board-like. There was marked direct and rebound tenderness in the epigastrium. Rectal examination was negative. He weighed 116 lb.

Urinalysis was negative. The hematocrit was 45%. Hemoglobin was 13 gm. per 100 cc.; the white cell count was 14,300, with 35% polymorphonuclears and 61% lymphocytes. A diagnosis of perforated peptic ulcer was made, and laparotomy was carried out. There was a small perforation in the anterosuperior portion of the duodenal bulb. This was closed with a tag of omentum by the Roscoe Graham technique. The patient's postoperative course was uneventful, and he was placed on a high-caloric, high-protein diet.

On Feb. 19, 1954, subtotal gastrectomy, gastrocoloduodenostomy, and transverse colocolostomy

Fig. 5 (Patient A. P.).—Roentgenogram of March 16, 1954, shows a normal-appearing segment of colon placed between the gastric remnant and the duodenum. There is no evidence of dilatation or atony.

and appendectomy were carried out. A segment of transverse colon 20 cm. in length was intercalated to form the neostomach.

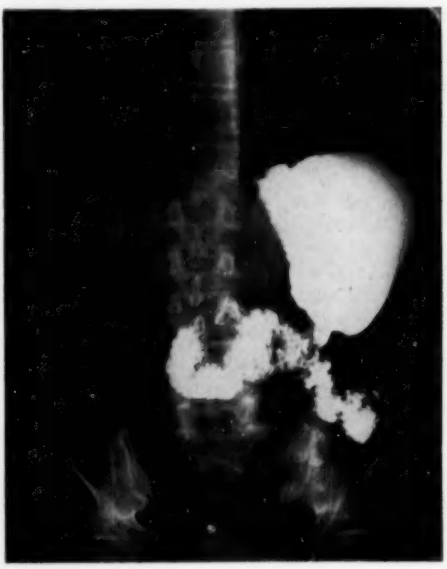
The patient's postoperative course was uneventful, and he was discharged on March 10, 1954. An upper gastrointestinal series done on March 16, 1954, showed a normal-appearing segment of colon placed between the gastric remnant and the duodenum. There was no evidence of dilatation or atony (Fig. 5).

He was readmitted on Aug. 9, 1954, for evaluation. After an uneventful convalescence from his operation he had noted occasional gnawing midabdominal pain which came on two hours after eating and was relieved by food. There was no flatulence, nausea, or vomiting. He had gained approximately 7 lb. in the past six months. Aside from chronic asthma he had no other complaints. The hemoglobin was 11.5 gm. per 100 cc., the leucocyte count was 11,500 with 84% polymorphonuclears. Urinalysis was negative except for an occasional leucocyte. The total protein was 6.3 gm. per 100 cc., with 4 gm. of albumin. An upper gastrointestinal series revealed a subtotal gastric resection with replacement of the distal end of the stomach by a section colon. A gastrointestinal series done on July 14 showed no obstruction of an organic nature or ulceration, but there had been definite retardation of the movement of the barium into the duodenum. The gastric remnant was dilated (Fig. 6). The gastric mucosa appeared normal on gastrostomy. The colon transplant had the same flat pink-brown color previously seen. No ulcer was noted, and no peristaltic movements were seen in the colon.

Immediately after the operation he had gained 5 lb. However, by June 29, 1954, he had lost 5 lb. and complained of aching hypogastric pain which was aggravated by cold water. On June 8, 1954, the patient had an acute exacerbation of bronchial asthma. He was seen in the surgery clinic July 7, at which time his weight was 112 lb. He complained that several hours after eating he would get an aching abdominal pain relieved by food. There was no night distress or nausea or vomiting. He stated he had to eat small volumes, but he complained of no sweating or symptoms of dumping. On July 21 his weight was 113 lb. He was seen again on Oct. 21, at which time he was suffering from an acute asthmatic bronchitis, precipitated by an upper respiratory infection. His weight at this time was 119 lb., and he complained of no gastrointestinal symptoms.

Comment. — Roentgenologic examination of the neostomach showed the characteristic slow emptying time. Despite his acute exacerbations of asthmatic bronchitis, he was able to maintain his weight.

Fig. 6 (Patient A. P.).—Roentgenogram of July 14, 1954, shows no obstruction of an organic nature or ulceration, but there is definite retardation of the movement of barium into the duodenum. The gastric remnant is dilated.



In this case there had been a functional obstruction to the passage of barium from the gastric pouch into the colon segment; this had developed between March and July and had gradually subsided.

CASE 4.—E. B., a 70-year-old arthritic white housewife, was admitted to the Denver General Hospital on March 4, 1954. She had had her first ulcer symptoms in 1917. For the past 12 years she had had a known duodenal ulcer verified by roentgenologic examination. During this interval of time she had had five episodes of melena and occasional hematemeses. On March 1 she noted black tarry stools, and on March 2 she vomited 1 pt. of dark liquid material.

Her usual weight had been 125 lb; her weight on admission was 116 lb. She had lost 10 lb. in the last five months.

Physical examination revealed a well developed, well nourished white woman, very pale, lying in bed but not complaining. She was alert, conscious, and cooperative. Skin was sallow, warm, and dry. She was edentulous but wore plates. Her blood pressure was 145/80; pulse was 88; respirations were 20. Normal heart sounds were present. There was a lower abdominal midline well-healed scar. Black tarry feces were found on examining finger in rectum. Arthritis of hands and knees was found.

On admission the hematocrit was 34%. The hemoglobin was 11 gm. per 100 cc. Total leucocytes numbered 9400, with 79% polymorphonuclears. Urinalysis showed a few erythrocytes and crystals. The blood nonprotein nitrogen was 66 mg. per 100 cc. The feces were positive for occult blood. The total protein was 5.9 gm. per 100 cc., with albumin of 3.72 gm. and globulin of 2.18 gm. The albumin-globulin ratio was 1.71. Electrocardiogram showed an intraventricular block and evidences of an old infarct. The patient was admitted to the medical service, where she received 4 units of whole blood, with much improvement. She was placed on an ulcer regimen. On March 5, 1954, her hemoglobin was 11 gm. per 100 cc., but she apparently continued to bleed, and her hemoglobin later dropped to 7.5 gm. on March 18, 1954. On March 9, 1954, a gastrointestinal series revealed a normal esophagus, a small sliding hiatus hernia, and normal stomach, but a severely deformed duodenal bulb.

The patient was transferred to the surgical service on March 13, 1954. Operation was undertaken on March 19, 1954. The duodenum was found to be scarred and bound down. It was impossible to mobilize enough duodenum for an end-to-end anastomosis; consequently the duodenal stump was closed, the anterior duodenal wall and the pancreatic capsule being used. Approximately 75% and the lesser curvature of the stomach were resected. A piece of transverse colon approximately 75 cm. long was then obtained. The second part of the duodenum was freed by incising the peritoneum lateral to it and the proximal end of the colon segment was approximated to it, so that an end-to-side anastomosis was performed. The distal end of the colon segment was then anastomosed to the gastric stoma.

Her postoperative course was uneventful. After operation she had no complaints.

An upper gastrointestinal series was carried out on April 8, 1954. It showed the barium meal to traverse the esophagus normally. It entered the remainder of the gastric pouch and flowed rapidly into the transplanted portion of the colon, which extended from the inferior extremity of the remaining stomach straight down to the brim of the pelvis, where it turned sharply to the right and ascended to its junction with the apparent location of the duodenum at the level of the second lumbar vertebra. Residual barium from the previous examination outlined the colon well on this examination, so that the two structures could be differentiated from each other. During fluoroscopy, it was noted that the colon transplant went into spasm, which caused the patient some degree of pain and also caused regurgitation of the barium meal from this portion of the colon into the remaining gastric pouch. Roentgenograms failed to show any evidence of the barium meal passing into the small intestine even after five hours. A gastrointestinal series showed some residual barium at one and four hours in the neostomach. However, barium was present in the small bowel at four hours. She had no gastrointestinal complaints and weighed 118 lb. (preoperative weight was 115 lb.).

On Aug. 11, 1954, a gastric analysis showed no free acid and 34 degrees of total acid. Gastroscopy showed a normal gastric mucosa. The colon mucosa appeared to have the same pinkish-brown color as previously noted.

Comment.—In spite of the fact that this patient had a transverse colon implant of 75 cm. length and in spite of the slow emptying time of the neostomach, this woman had the smoothest convalescence of all those of our series. She complained of abdominal cramps following the ingestion of food only during the second postoperative week.

CASE 5.—P. C., a 66-year-old Spanish-American man, was admitted to the Denver General Hospital on Dec. 24, 1952, because of abdominal pain. The pain had begun on the morning of admission following breakfast. It began in the right upper quadrant and was mild at the onset and not colicky. Within an hour it localized in the epigastrium where it remained; occasionally it radiated through to the back.

The patient's general health had been good. He had been followed in the chest clinic for seven years because of findings on the chest roentgenogram of old, healed tuberculosis; he had shown no change radiographically and had never had any sputum.

Examination showed a well-developed, well-nourished man who was acutely ill and lying upon his right side. His blood pressure was 165/100; pulse was 108; his temperature was 38.2 C (100.8 F), and respirations were 20. The abdomen was slightly distended and rigid throughout. Marked tenderness was present in the right hypochondrium. The hemoglobin was 15 gm. per 100 cc. The total leucocytes numbered 16,200, with 96% polymorphonuclear neutrophiles. The serum amylase was 292 units. Roentgenological examination of the abdomen showed the absence of free air in the peritoneal cavity.

Laparotomy on Dec. 25, 1952, confirmed the clinical diagnosis of acute pancreatitis. A choledochostomy was made and T-tube drainage instituted. His course was satisfactory, and he was discharged on Jan. 12, 1953.

He was again admitted to the hospital, on Feb. 13, 1954, because of acute upper abdominal pain of six hours' duration. The pain radiated to the interscapular region. Examination demonstrated that he was in mild acute distress. His blood pressure was 160/100 and the pulse was 114. Respirations were 30. His temperature was 38 C (100.4 F). The abdomen was distended, and there was marked tenderness in the right epigastrium.

The hemoglobin was 17 gm. per 100 cc. Total leucocytes numbered 16,300, with 95% polymorphonuclear neutrophiles. The hematocrit was 61%. Urinalysis was negative. Serum amylase was 225 units.

He was treated with gastric suction, atropine, meperidine (Demerol), and repletion therapy. This treatment was continued until Feb. 24, 1954, when a cholecystogram was attempted, but the visualization was inadequate for diagnostic purposes. Free hydrochloric acid was present in the aspirated stomach contents. An upper gastrointestinal series on March 15, 1954, showed an ill-defined lesion in the greater curvature of the stomach in the region of the fundus which was interpreted as an intrinsic invasive lesion of the stomach or an extrinsic mass infiltrating the stomach in this area. A film taken several hours after the upper gastrointestinal series showed that most of the barium was retained in the stomach. The diagnosis of complete obstruction at the pylorus was made. A chest roentgenogram showed fibrocalcific densities present within both upper lobes. There was thickening of the apical pleura bilaterally, unchanged since prior examination.

Because of the ulcerating gastric lesion and the pyloric obstruction, operation was advised and was undertaken on April 2, 1954. Exploration of the peritoneal cavity revealed a markedly indurated head of the pancreas with the scar extending into the proximal duodenum and a well-circumscribed ulcer of the greater curvature of the stomach, somewhat on the anterior surface of the stomach, which measured 2 cm. in diameter. Gastric resection was carried out.

Frozen section examination of the removed tissues showed that the gastric lesion was an extremely anaplastic ulcerating adenocarcinoma. The removed omentum showed fat necrosis.

On April 9, 1954, he developed profuse drainage from the left Penrose drain. Dye taken orally was seen to be on the dressings. He was placed on nothing by mouth and parenteral alimentation. On May 8, 1954, after having been on all feedings without difficulty for two weeks, he began to drain again. A Foley catheter was placed in the gastrocutaneous fistula, and feedings were continued. On May 22, it was noted that he was improving slowly but vomited occasionally. On June 5, 1954, he had only slight drainage from his abdominal wound. His condition was slowly improving. On June 29, 1954, he weighed 93 lb.; he had gained 3 lb. since discharge from the hospital. On July 20, 1954, his weight was 91½ lb., and there was a nodular mass in the right upper quadrant. No free acid was present in the gastric aspirate. The provocative pepsinogen test gave a resting value of 50 tyrosine units, and stimulation failed to demonstrate any increases over a four-hour period.

Comment.—The chances of primary healing were jeopardized here by the presence of a subacute pancreatitis. The gastrocutaneous fistula responded well to conservative management. His failure to gain weight may be related to the development of a nodular, apparently neoplastic mass in the right upper quadrant.

CASE 6.—J. J. S., a 55-year-old white alcoholic cab driver, had been well until November, 1953, when he began to have typical epigastric ulcer pain. On Dec. 17, 1953, he first presented himself at the hospital and was placed on a medical regimen. On Feb. 23, 1954, he fainted, developed severe epigastric pain, was seen again in the emergency room and further instructed regarding medical therapy. This episode was associated with black tarry stools. He had three separate attacks of melena subsequently. On March 9, 1954, he again began passing black tarry stools, noticed some weakness, and complained of dyspnea at rest. He had lost 15 lb. of weight in the previous three months.

Fig. 7 (Patient J. J. S.).—Roentgenogram of April 16, 1954, shows the progress of a barium meal three and three-fourths hours after ingestion. Some of the barium is in the small bowel, but most of it still remains in the neostomach.

Examination showed a well-developed, well-nourished, florid-faced white man, who did not appear ill. The blood pressure was 130/100; the pulse was 100; the temperature was 37 C (98.6 F). He weighed 135 lb. He had a pugilistic deformity of the right pinna. The mouth contained many carious teeth and pyorrhea was severe. Heart sounds were normal, and the lung fields were clear. The abdomen was scaphoid and soft; there was slight tenderness in the midepigastrium.

On March 9, 1954, examination of the blood showed that the hemoglobin was 13.5 gm. per 100 cc., with 45% hematocrit. The white cell count was 7,500, with 66% polymorphonuclears. Urinalysis was negative. Total blood protein was 6.9 gm. per 100 cc., with 4.8 gm. albumin. Stools were positive for blood.

Roentgenographic examination showed large rugae without evidence of ulceration or filling defect in the stomach. The distal portion of the duodenal bulb had a pinched appearance, suggesting either a scar deformity or spasm. No ulcer crater was visualized.

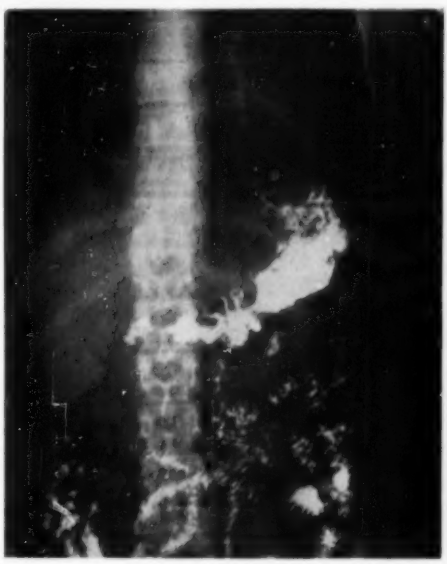
Operation was carried out on March 23, 1954, and a chronic fibrotic duodenal ulcer was found. Gastric resection was carried out, with removal of 70% of the stomach.

On the third postoperative day large amounts of bile-stained fluid were noted through the drain site, and methylene blue administered by mouth appeared in the drainage. A Levin tube was replaced, and a sump drain connected to a Stedman pump was placed in the stab wound. On April 15, 1954, the patient was discharged with his abdominal wound entirely dry.

An upper gastrointestinal series on April 16, 1954, showed the progress of a barium meal three and three-fourths hours after ingestion. Some of the barium was in the small bowel, but most of it still remained in the neostomach (Fig. 7).

Immediately after his operation he had a very poor appetite and had occasional vomiting, usually six to eight hours after eating. This had occurred not more than twice weekly, and he had gone for periods exceeding a month without vomiting. His weight at the time of discharge was 119 lb.; on Aug. 6, 1954, his weight was 120 lb. Gastric analysis showed no free acid. Stools were negative for occult blood. Total protein was 7.7 gm. per 100 cc., with 4 gm. of albumin. An upper gastrointestinal series showed the esophagus and remaining portion of the stomach to be normal (Fig. 8). The films taken at one and four hour intervals showed residual barium of a moderate amount (Fig. 9). The provocative pepsinogen test showed resting values of 50 tyrosine units and no elevations from stimulation over a period of four hours. He was last seen on Aug. 31, 1954, when his weight was 126 lb. He had gained 6 lb. since his previous visit.

Fig. 8 (Patient J. J. S.).—Roentgenogram of Aug. 11, 1954, was made one-half hour following the ingestion of a barium meal.



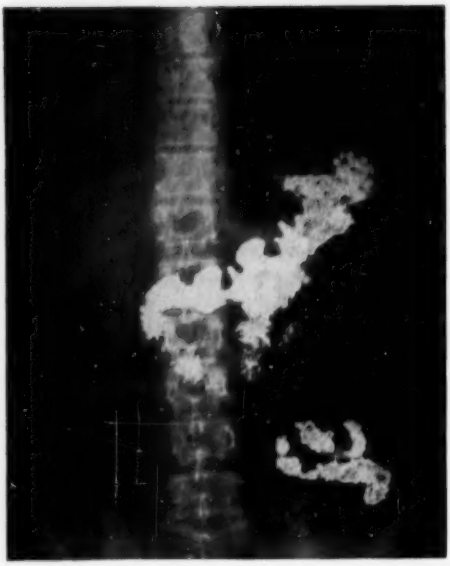


Fig. 9 (Patient J. J. S.).—Roentgenogram of Aug. 11, 1954, was made four hours after the barium meal. A considerable amount of barium still remains in the neostomach.

Case 7.—C. H., a 61-year-old white man pensioner, had been told in 1930 that he had a duodenal ulcer. In January, 1952, a roentgenographic examination of the stomach demonstrated a duodenal ulcer. He was placed on a medical ulcer regimen. He remained well until December, 1953, when he came to the Denver General Hospital because of the pain in the left hypochondrium, which was always relieved by food. His appetite was poor.

His weight estimations were as follows: In January, 1952, he weighed 167 lb.; in January, 1953, 119 lb.; in December, 1953, 157 lb., and in March, 1954, he weighed only 137 lb.

Physical examination demonstrated a thin, tall, white, 70-year-old man who was cheerful and without complaints. His blood pressure was 238/118. The pulse was 66; respirations were 16. He was 6 ft. tall and weighed 137 lb. The ocular fundi showed marked narrowing of the arteries with prominent nicking of the veins. There was evidence of old chorioretinitis on the left. He was edentulous. The left maxillary sinus opened into the superior labial fold; there was no evidence of any drainage. The maximum left border of the heart was at the sixth interspace on the anterior axillary line. The abdomen was flat; no rigidity was present; there was a tender area 2 or 3 fingerbreadths to the left of the midline below the costal margin extending to the midline.

Examination of the blood showed the hemoglobin to be 13 gm. per 100 cc.; the leucocytes numbered 10,200 with 68% polymorphonuclears. The urine showed a few hyaline casts and a few leucocytes. The total blood proteins were 7.5 gm. per 100 cc., with 4.8 gm. of albumin. The stools were negative for occult blood. The electrocardiogram showed premature ventricular contractions. Left ventricular hypertrophy was present, and the heart was in the horizontal position. A gastrointestinal series showed evidences of a prepyloric ulcer (Fig. 10).

Operation was carried out on April 15, 1954. The peritoneum was opened and the abdomen was explored. Not only the prepyloric ulcer but also a smaller ulcer at approximately the middle of the lesser curvature, both of which proved to be benign, were found. A 75% gastric resection was accomplished with implantation of a segment of transverse colon measuring approximately 35 cm.

Postoperatively the patient developed an exacerbation of a chronic left maxillary sinusitis. His course was marked by episodes of distention and considerable abdominal, cramping pain. A gastrointestinal series on May 10, 1954, showed that the barium passed freely into the gastric pouch. However, no peristalsis or motion of the colon pouch was noted, and barium remained in it for as long as three hours. At that time some barium was seen further on in the small bowel and the examination was discontinued. Although no stenosis was observed, there was fluid retention before the exam-

Fig. 10 (Patient C. H.).—Roentgenogram of March 9, 1954, shows evidences of a prepyloric ulcer.

ination was started and retention of barium in the neostomach as long as three hours with little passage into the small bowel. The retention appeared to be due mostly to the lack of peristaltic activity in the transplant rather than to obstruction. He continued to have episodes of cramping epigastric pain and distention, with occasional regurgitation; these were precipitated by cold ice cream or milk.

On Aug. 5, 1954, three and one-half months after operation, he complained of failure to gain weight and abdominal cramps after eating. He was unable to eat large amounts of anything and regurgitated at least once a day. His bowels moved every day and were normal in color. He weighed approxi-

Fig. 11 (Patient C. H.).—Roentgenogram of Aug. 10, 1954, shows the neostomach immediately after ingestion of a barium meal. Some barium remained in the pouch after 4 hours.

mately 119 lb. when he left the hospital. His weight on August 5 was 119 lb.; normal weight was 145 lb.

Examination showed a pulse of 80 and the blood pressure was 170/100. He appeared malnourished and anemic. Hemoglobin was 14 gm. per 100 cc.; leucocytes numbered 9,000, with 60% polymorphonuclears. Urinalysis showed an occasional leucocyte. Gastric analysis showed 10 degrees of free acid. An upper gastrointestinal series on Aug. 10, 1954, showed that there were tertiary contractions of the esophagus but no organic pathology. Barium passed into the gastric remnant, which showed evidence of retention of food or fluid from the previous night. It then passed into the J-shaped colonic transplant without obstruction. However, the barium meal remained in this with very little passage into the jejunum four hours after the original meal. There was no evidence of organic obstruction, but there was retention of barium within the colon transplant (Fig. 11). A 10-hour film still showed a considerable residual of barium within the gastric remnant and colon transplant. A flat plate of the abdomen on Aug. 12, 1954, showed that the barium administered 24 hours previously had left the stomach and the stomach prosthesis and was then in the terminal ileum and colon. Gastroscopy was carried out on Aug. 13, 1954; all portions of the stomach and neostomach were well visualized, and no abnormality was seen. Ten degrees of free acid was found in the stomach aspirate.

The provocative pepsinogen test showed very low resting values and no augmentation on stimulation over a period of four hours.

He was seen again in the surgery clinic on Aug. 25, 1954, at which time his weight was 128½ lb. During July he had continued to have occasional difficulty with regurgitation. He was seen again on Sept. 27, 1954, at which time he weighed 128 lb. His appetite was good and he had normal bowel movements. His main complaint was delayed emptying of the stomach and occasional regurgitation of foul-smelling stomach contents. He was continued on belladonna, phenobarbital, and psyllium hydrophilic mucilloid with dextrose (Metamucil), which gave symptomatic relief.

Comment.—The postoperative symptoms were dominated by intermittent attacks of cramping epigastric pain and regurgitation of foul-smelling stomach content. In the absence of any evidence of obstruction this must be attributed to atony or spasticity of the colon implant. The latter possibility is indicated because of the marked symptomatic relief produced by atropine.

GENERAL OBSERVATIONS

We have performed subtotal gastrectomy with reconstruction according to the Billroth I plan, utilizing segments of transverse colon to join the gastric stoma to the duodenum. The indications for operation have been the same as for any type of gastric resection. In all our cases this procedure has been one of election, and the colon has been prepared. We do not believe one would be justified in attempting this type of recon-

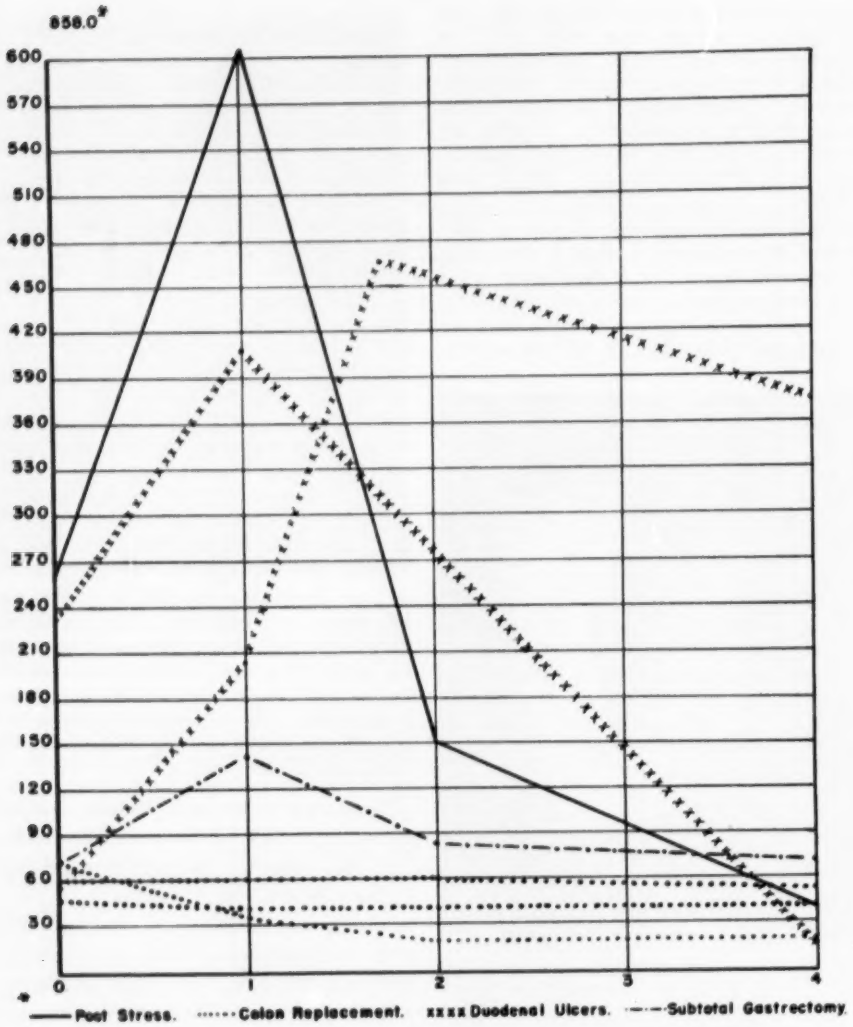


Fig. 12.—Results of the provocative pepsinogen test. The ordinates represent tyrosine units; the abscissae represent time in hours. The post-stress curve (solid black) was obtained after the administration of corticotropin (ACTH) intravenously. The value 858 is the highest value we have recorded; it was obtained from a patient after craniotomy for aneurysm. He died of bleeding duodenal ulcer. Note the low values obtained in the colon implant cases after gastric resection.

struction in cases of emergency gastrectomy with an unprepared colon.

The assumption has been made that the unfortunate sequelae of gastrectomy reside in the method of reconstruction. In view of the numerous procedures now being evaluated in the search for a better "ulcer operation," we felt justified in attempting the reconstruction proposed here.

Whether a colon implant is intercalated between the esophagus and the duodenum in the case of total gastrectomy or between the gastric remnant stoma and the duodenum in the case of subtotal distal gastrectomy, in

our experience all types of colon segment implants are associated with transient postoperative disturbances of motility. Usually the early convalescence following a Hofmeister-Polya type of operation is more satisfactory than the early response of the Billroth I. There is no retention or distention of the gastric remnant. However, as time passes, the patient with the Polya type of operation is more likely to develop symptoms of the so-called postgastrectomy syndrome. In our cases the early postprandial cramps have usually rather promptly subsided, with limitation of fluids taken at mealtimes and with the administration of antispasmodics. The radiological appearances of these disturbances in motility are illustrated by Figures 3, 4, 5, and 6. In general, the course has been one of steady, continuous improvement. However, some patients have continued to be unable to ingest certain specific substances, especially very hot or cold liquids, without discomfort.

Regurgitation of undigested food and bile which is seen occasionally as an intermittent occurrence after total gastrectomy with colonic replacement has not been observed in this group, presumably because of preservation of the cardia.

No instances of the usual postgastrectomy syndrome have been found.

The examinations of the aspirates from the neostomachs have shown anacidity or very low values for free acid. The fluid is generally very mucoid; aspirates are all obtained in the morning from the fasting viscus. We have been using a method for assessing the ability of the stomach mucosa to produce pepsin in response to a stimulus as an index of treatment and prognosis. This method we have called the provocative pepsinogen test; it will be reported in detail elsewhere. Illustrative cases are shown in Figure 12. The low values obtained in three cases of subtotal gastrectomy with transverse colon implant compared to the value obtained from a typical subtotal gastrectomy are illustrated. Zero values are obtained with this test in cases of total gastrectomy.

The resistance of the colonic mucosa to the secretions produced by the residual gastric remnant within the time intervals studied has been well demonstrated by gastroscopic observations in addition to the roentgenologic examination. No abnormalities of the mucosa were noted.

The evaluation of this procedure must await time and a broader basis of observation.

SUMMARY

Seven cases are presented in which subtotal gastrectomy was performed and restoration of continuity was accomplished by the intercalation of a segment of transverse colon between the gastric remnant and the duodenum.

Salient features of these cases are compared.

Subtotal gastric resection (in the cases of duodenal ulcer) of 75% to 80% is an essential preliminary to decrease gastric hypersecretion.

The use of a segment of transverse colon makes possible a Billroth I type of reconstruction subsequent to gastric resection. The segment of transverse colon is ideally adapted anatomically to restore the crop function of the stomach. Food is held in the neostomach and delivered slowly to the duodenum.

The immediate results suggest that the preservation of total duodenal function as an active factor in digestion is worthwhile.

In our experience all types of colon segment implants are associated with transient postoperative disturbances based on altered motility of the segment.

No instances of the postgastrectomy syndrome ("dumping") have been encountered.

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A Modified Valvular Gastrostomy

RICHARD PATTON, M.D., Columbus, Ohio

A great many varieties of gastrostomy have been proposed and many variations are described in texts on surgical technique. Although many successful procedures are available to the surgeon, each has its disadvantages, and the ideal permanent gastrostomy has not been described. A technique of gastrostomy will be presented which, although not ideal, has been a satisfactory permanent gastrostomy.

The ideal gastrostomy should be simple to perform and easily tolerated by the patient. It should require no permanent tube, and the feeding tube should pass with ease so that the patient can feed himself. The gastrostomy stoma should not seep or leak and should require a minimum of after-care. The chief indication for gastrostomy is advanced malignant disease of the gastrointestinal tract above the stomach. In this type of patient the gastrostomy should be permanent for a life expectancy of a number of months and is not uncommon.

In spite of the great number of gastrostomy procedures that have been described, there are basically two types of gastrostomy. The first is the serous tract gastrostomy, in which the stomach wall is infolded and a tube is placed in this infolded wall and brought out through the left upper quadrant. The tract in this case is lined with serosa. This type of gastrostomy is represented by the Stamn, the Senn, and the Witzel operations. These procedures are simple technically and are well tolerated by the patient and are effective as long as the permanent tube is left in the stomach. They are completely satisfactory as a temporary gastrostomy. In this series they have not been satisfactory as a permanent gastrostomy. Attempts have been made to modify these three procedures in such a way as to produce a permanent gastrostomy with intermittent feeding. In general, the result has been that if the serous-lined tract is short and straight, as in the Stamn or Senn procedure, there is leakage when the tube is removed. If the tract is made very long, as in the Witzel, there is a strong tendency for the tract to close even with intermittent feeding and passage of the gastrostomy tube three or four times daily.

The second type of gastrostomy is the mucus-lined gastrostomy, as represented by the Janeway and the Spivak procedure. In these procedures a full-thickness flap of gastric wall is raised and the tube formed of this flap is brought out through the abdominal wall so that a mucus-lined fistula leads from the anterior abdominal wall to the interior of the stomach. If the original Janeway procedure is modified by forming a valve of full-thickness gastric wall at the base of the tube, leakage of gastric contents of the fistula can usually be prevented. This type of gastrostomy has many advantages. Although it is somewhat complicated to perform, it is easily tolerated by the patient and requires no permanent tube. The feeding tube passes with ease. This type of gastrostomy has one serious disadvantage which has occurred in all cases in this series. This disadvantage is a continual seepage which occurs from the mucous tract and causes skin excoriation around the fistula. The seepage apparently comes from the mucosa lining the tract. In spite of careful attention to the wound the patient has complained of continual seepage and skin excoriation. The problem has not been solved by cautery of the tract in our cases.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Dec. 3, 1954.

This modification of the valvular gastrostomy is designed to obviate the seepage and skin excoriation. Operations have been performed in six cases, the first in August, 1951. This modified valvular gastrostomy is technically slightly more difficult than the usual valvular gastrostomy but is easily tolerated by the patient, and in all cases has been performed under local infiltration anesthesia with 1% procaine. No permanent tube has been required, and seepage and leakage have not been problems. A minimum of after-care has been necessary, and the gastrostomy tube has been removed between feedings on the seventh postoperative day. Most patients have been able to feed themselves, and in most cases the passage of the feeding tube has been easy. A partial closure of the tract requiring dilatation occurred in one patient. This patient had malignant disease of the pharynx and upper esophagus and regained his ability to eat by mouth eight weeks after the gastrostomy had been performed. He went for two and a half weeks without using his gastrostomy for feeding purposes but at the end of that time required tube feeding again. The tract was successfully dilated with cervical dilators and the catheter was left in place for five days, and at the end of that time the usual intermittent feedings were resumed without difficulty. No further dilatations were required after the patient resumed feedings. The patient lived for 10 months and tube feedings were continued during that time. The problem of closure of the gastrostomy tract has not been observed in those patients who have continued frequent feeding without interruption.

From observations on a series of 30 gastrostomies of various types it is felt that for a temporary gastrostomy, a Stamn or Senn operation with a permanent tube is the procedure of choice. It is simple and entirely adequate. The permanent tube is a disadvantage if the gastrostomy is to be used for over a few weeks. The absence of a permanent tube and the advantages of intermittent tube feeding more than compensate for the increased technical difficulty of a valvular gastrostomy. The short postoperative course, satisfactory function of the valve, and absence of seepage from the tract support this modified valvular gastrostomy as a satisfactory permanent gastrostomy.

TECHNIQUE

Local infiltration anesthesia and an upper left rectus incision have been used in all cases. The gastrostomy tract has been brought out through a stab wound made just to the left of the upper left rectus incision. The stomach is mobilized, and a square 4 cm. on the side is marked out on the anterior gastric wall in the lower part of the middle third of the stomach midway between the curvatures. An incision through the serosa and muscular layer is made along three sides of this square, leaving the fourth side along the greater curvature to serve as the base of the flap. A dissection plane is developed between the mucosa and the muscle layer, and the flap is raised from the mucosa and left attached along the greater curvature. Bleeding is controlled by individual ligation of the vessels. The dissection plane is above the submucous plexus of vessels so that the major part of the blood supply is left on the mucosal side (Fig. 1). The 4-cm.-square area of mucosa which is left behind after the flap has been elevated is used to form the valve. The interior of the stomach is entered by incising the mucosa along the side of the square close to the lesser curvature. This incision through the mucosa is then carried along the superior and inferior sides of the square to the half way point (P and P,[1] Fig. 2). This forms a flap 4 cm. wide by 2 cm. deep which is folded outward and comes to rest on the other half of the original 4 cm. square of mucosa. It is sutured in place with 0000 nonabsorbable (silk) sutures. This forms a fold of gastric mucosa 4 cm. wide and 2 cm. deep which serves as the valve. The opening of the gastric wall is closed transversely over a catheter (Fig. 3). Interrupted sutures through the full thickness of the gastric wall are taken so as to approximate the superior and inferior sides of the square. A single suture line transverse to the long axis of the stomach is

produced. The first suture is taken at point *X* (Fig. 2), and *A* and *A*,[1] *P* and *P*,[1] *B* and *B*[1] and *C* and *C*[1] are joined (Figs. 2 and 3). A second layer of inverting seromuscular sutures is taken to support the suture line. These suture lines complete the valve and form the seromuscular tract around the catheter which is brought out through a stab wound just to the left of the incision. The anterior gastric wall is sutured to peritoneum around the stab wound and the incision is closed.

POSTOPERATIVE CARE

Continuous suction is usually applied to the gastrostomy tube for about 12 hours, at the end of which time small amounts of water are started through the tube. If the water is well tolerated, the usual feeding formula is administered after 24 to 36 hours. After seven days the tube is removed and inserted four to five times daily for tube feedings. Leakage has not been a problem, and after the feeding has been accomplished the tube is removed and a small dressing applied.

Tube feedings must be performed at least daily. If tube feedings become unnecessary, the catheter should be inserted daily to prevent closure of the tract.

LABORATORY WORK

In proposing a technique involving splitting the gastric wall to form a valve and a flap, it is necessary to know what happens to each segment of the split wall. In the animal laboratory the modified valvular gastrostomy as described above was performed on three dogs. Difficulties were encountered in keeping the gastrostomy tube in place for the seven-day period desired. In spite of plaster body casts the dogs tore out the tubes in three days. Rubber and metal catheters were inserted intermittently to simulate feedings. There was a strong tendency of these tracts to close in the dog, and if even a few days elapsed between insertion of the catheters passage was difficult. This was much more pronounced in the dog than has been noted in the patient. The dogs were killed after seven, eight, and nine weeks. The stomach and abdominal wall were removed together. They were examined and photographed and microscopic sections were taken of the valve and the tract.

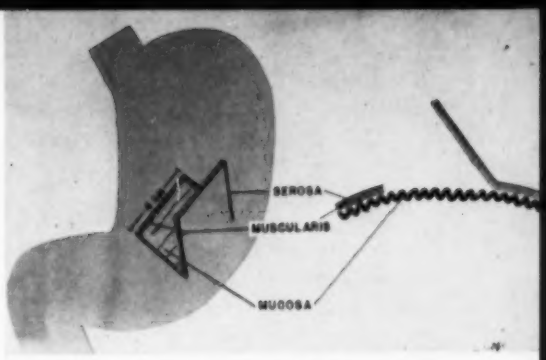


Fig. 1.—Formation of flap.

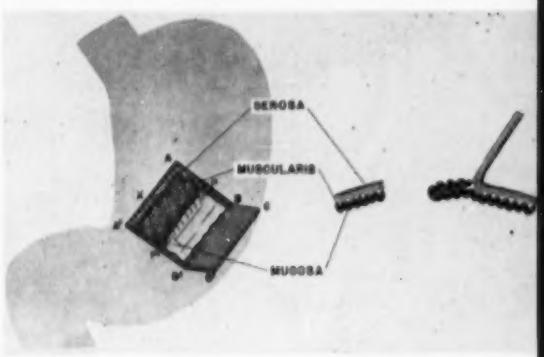
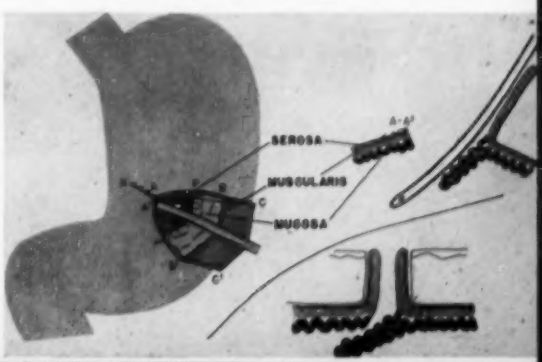


Fig. 2.—Formation of valve.

The valve of mucosa had remained viable and soft. It worked on the principle of the flutter valve, the two leaves falling together with the stomach empty or distended. The valve did not leak when the stomach was distended with water, although a 16 F. rubber catheter could be passed through the tract with ease from either direction.

Microscopically, the muscle layer of the tract was viable but no mucosa had formed. The tract is basically a fibrous tract, but the muscle and serosa in the wall form a firm fistula for passage of the feeding tube.

Fig. 3.—Formation of tract.



SUMMARY

A modified valvular gastrostomy has been described. It is well tolerated by the patient, does not seep or leak, and requires no permanent tube. It does not meet the requirements of an ideal gastrostomy in that it is somewhat complicated to perform and requires daily tube insertion if tube feedings are not being given. However it has been a satisfactory permanent gastrostomy in six cases.

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DISCUSSION

OF PAPERS BY DRS. WATKINS AND WITTENSTEIN AND PATTON

DR. FRED R. HARPER, Denver: A number of years ago (as a matter of fact, too many years ago)—in 1931, to be exact—I was working in the laboratory of Dr. Frank Mann. The experiment I worked on produced peptic ulcers.

[Slide] The operation is done in two stages. The first stage: a loop of intestine is anastomosed to the stomach and the gastrointestinal continuity is reestablished. After that, nothing happens. Some time later a Heidenhain pouch is made, including the intestinal segments.

The result of this procedure is that as soon as the Heidenhain pouch is made you get in 100% of experimental animals ulcers developing in the intestinal segments. I had numerous studies of secretions to go with it, but if you want to form ulcers on an experimental basis this is an excellent method. It will give you 100% ulcers.

[Slide] If you use a loop of ileum, the ulcers will form very rapidly; as a matter of fact, they will form in from seven to eight days and will perforate.

[Slide] On the other hand, if you use a loop of jejunum or duodenum, the ulcers form very slowly and are chronic. These ulcers generally do not perforate.

As a result of these experiments, I concluded that the mucous membrane of the intestine becomes more susceptible to gastric secretion as you go further away from the stomach.

In some of the dogs, if a Pezzer catheter was put in the pouch at the completion of the second stage of the operation so that it drained freely, ulcers did not develop until the catheter was removed and then ulceration would develop. As long as there was free drainage from the Heidenhain pouch, ulceration did not occur, which may have something to do with Dr. Watkins' operation.

I would have assumed, and so told Dr. Watkins when he first did these operations, that he would get ulcerations if he used the colon, because the colon would be very susceptible to acid secretion. However, that has not been the case in his cases, and I know his patients and know that they have not had ulceration.

The explanation is probably that he gets adequate drainage, or possibly, since we did not use the colon, the colon is resistant to gastric secretion. As a little experiment I think it might be worthwhile to prepare a few of these dogs with colon and see if the colon actually is resistant to gastric secretion; if it is, it certainly would be an excellent mucous membrane to use in reestablishing continuity of the stomach.

DR. CLAUDE J. HUNT, Kansas City, Mo.: I am interested in this paper by Dr. Watkins. It is a very ingenious procedure to replace a segment of the stomach by a portion of the gastrointestinal tract. It shows very definitely that medicine is never static and that we are always trying to improve upon the known and tried.

Henley, of England, has done something similar by making a neostomach out of a segment of jejunum. These procedures are intended to maintain the normal flow of gastric contents by way of the duodenum, to combat nutritional deficiency, and to eliminate some other undesirable results of subtotal gastrectomy which are occasionally encountered. These efforts are commendable, and those who do them are to be congratulated.

We have been replacing the stomach by segments of the gastrointestinal tract for a good many years, and we have presented the results to this Association on one or two occasions. We have never been greatly enthusiastic about taking a segment of the colon and replacing the stomach by the right colon or the transverse colon.

I know that Dr. Hunnicutt, of this Association, has explored this procedure and has done several operations on replacing the entire stomach by the right colon and has had good results.

Drs. State, Barcley, and associates, at the University of Minnesota, have used the transverse colon

to replace the stomach and have had excellent results by that method.

When you take a segment of the gastrointestinal tract from any place and transplant it into a new position, and use it, for example, as a substitute for the stomach, I don't know whether normal peristalsis is retained or not. If it does retain its peristalsis and you transplant the transverse colon and substitute it for the stomach, you have a reverse peristalsis.

As was shown in these pictures of the transplanted transverse colon, there seemed to be quite a delay in emptying of this segment of the neostomach, and the barium was retained as long as three hours, indicating that food contents would be retained for a similar period of time. This is similar to the retention seen when the entire stomach is replaced by a segment of the transverse colon. Perhaps reverse peristalsis has something to do with it.

The colon is not anatomically and physiologically adapted to receive the ingested food particles. The right colon is sacculated in character and has thin muscular walls. Any segment of the colon has always appeared to me to be an inappropriate portion of the gastrointestinal tract to transplant as a segment either in formation of a neostomach or for replacement of the entirely removed stomach.

We have used the most anatomical and the most adjacent part of the intestinal tract, the jejunum, as a substitute for the stomach.

We have made a double loop out of a jejunal segment, forming an enterostomy stoma between the parallel loops of the jejunal segment and have replaced the stomach by attaching this segment to the esophagus and the duodenum as Drs. Hensley and Longmeier have done with a single loop of jejunum. Jejunal continuity was restored by end-to-end anastomosis.

Our greatest success has been in making an artificial stomach or pouch out of the divided jejunum, doing a Roux-Y anastomosis of the proximal jejunal segment to the distal one and making a pouch out of the distal segment of the jejunum and attaching it to the esophagus.

We have used this method in nine cases. Eight have recovered satisfactorily from the operation and have lived various lengths of time, maintaining very good physical comfort.

The one operative hospital mortality resulting from a necrotic segment in the transverse colon occurred from the simultaneous disconnecting of a previously performed gastroenterostomy. The vascularity of the colon was unknowingly impaired; there was subsequent necrosis, resulting in an abdominal fistula with progressive decline of the patient and a demise in the ninth postoperative week. Autopsy showed a colonic jejunal abdominal fistula at the site of the Roux-Y anastomosis which diverted all oral intake into the distal colon and through the fistula.

Dr. Watkins and Dr. Wittenstein are to be congratulated upon their efforts to improve upon the surgical methods of gastric surgery.

DR. BERNARD G. LANNIN, St. Paul, Minn.: I want to discuss Dr. Watkins' paper and also answer Dr. Harper's implied question.

Dogs have been prepared with colon, Dr. Harper. Dr. Dragstedt did it a number of years ago, and I happened to have done it in Dr. Wangensteen's laboratory in about 1941. You are perfectly correct. The farther you go down the intestinal tract, the more susceptible it is to the action of acid juice, and the colon is worse than the ileum.

That is why I would caution that if you are going to replace a portion of the transverse colon, you must make sure that your gastric resection is adequate. If you do it with a small resection, I am sure you will get ulceration in that colon segment.

DR. JOHN GRINDLAY, Rochester, Minn.: Since the tone of the discussion so far is from the point of view of surgical physiology, I should first of all like to compliment Dr. Patton on his ingenious procedure, which I think shows that all the elements of surgical physiology have been considered.

In the case of Dr. Watkins' paper, the comments that have been made so far have raised certain questions. I should like to pose two other questions.

First of all, I wonder whether the fact that there is resistance to emptying of the stomach is not for the reason that after the ingestion of a meal, as you know, there is a gastrocolic reflex, i. e., a contraction of the colon soon after a meal. This is a reflex with which you are all familiar, a five-minute-after-breakfast reflex. Also, I wonder whether the roentgenograms which Dr. Watkins showed demonstrate only the phase of dilatation after a preceding contraction invoked by the gastrocolic reflex.

The other thing that struck me about his report was that this impediment cleared up after a few months. The colon of the human has no longitudinal muscle except the teniae. The dog, which has instead of teniae an incomplete layer of longitudinal muscle, is probably not the animal to use in such studies on the subject. In man, perhaps, the muscular arrangement of circular and longitudinal layers is such that a segment of colon in the position of the stomach is more like a gastric reservoir.

At any rate, I think Dr. Watkins is to be complimented for giving a long range follow-up of his patients after operation.

DR. ARTHUR JESSE HUNNICUTT, Oakland, Calif.: Although there are academic reasons why the colon should not be satisfactory in replacing the stomach, the clinical fact remains that it does work.

I think that we overlooked several fundamentals in our gastric resections, both total and subtotal.

First, that which we call the emptying time of the normal stomach is, I believe, largely a measurement of the acceptance rate of the duodenum. When we disrupt the so-called duodenopyloric reflex by surgical removal, there is no possible way that we can restore this relative control to the duodenum that it normally exercises over what it receives.

Second, the primary purpose in transposing a segment of bowel to replace all or a portion of the stomach is not to make a "food pouch" or to increase the size of the "food crop." I believe that the primary purpose is to slow down the stream of ingested food to a rate and consistency that is more readily acceptable to the duodenum. Most certainly, replacing the stomach with a segment of jejunum does not form a food pouch. Whether a segment of jejunum or colon or a loop of jejunum sewed upon itself is used this primary purpose of adjusting to the duodenum seems to be accomplished to some degree. Thus far, I believe that no one has sufficient proof that any one of these three variations in technique is superior to the other two.

The one question in my mind regarding this excellent presentation by Dr. Watkins is specifically on which patients he proposes to do this procedure. Let us remember that when we do a subtotal stomach resection for benign "peptic ulceration" we are recommending that the patient trade his organic disability for a surgical disability. We surgeons cannot make a person better than normal. We are, in effect, wagering that the surgical disability will be less than the patient's medical disability. Let us assume that we are correct in 80% to 90% of the cases with operation. Some of the remaining 10% to 20% may require further surgery. I presume that Dr. Watkins is suggesting the feasability of this procedure in selected cases in this latter group.

DR. DAVID H. WATKINS, Denver: Dr. Long, I should like to thank the Association for the privilege of presenting this work today, and I also would like to thank the discussers for their remarks.

You will note that I have refrained from making any recommendations or indications about this procedure which we have been interested in studying.

In the case of benign peptic ulcer, I am sure we would all agree that an effective pepsin inhibitor will probably be a more effective agent than a proposed operation.

In regard to the susceptibility of this tissue to gastric digestion, I should like to emphasize that in all of these cases a 75% to 80% gastric resection has been done, and during the course of this study when some of my colleagues asked me whether I was worried about dumping I stated that I was not worried about that feature at all. I was worried about whether these things would empty. They do, but it takes a period on the average of about four to four and a half hours.

In conclusion, I should like to emphasize that the delay in emptying of the neostomach may recommend its use in the treatment of so-called "dumping syndromes."

Nutritional Status Following Subtotal Gastrectomy with Jejunal Replacement

Experimental Evaluation

TILDEN C. EVERSON, M.D., Ph.D., Chicago
With the Technical Assistance of Everett Hoppe and Anna Poulos, B.S.

In recent years (1952) Henley [1] has suggested that the postgastrectomy nutritional disturbances which frequently occur after the Billroth II type of subtotal gastrectomy can be minimized if a jejunal segment is interposed isoperistaltically between the residual stomach and the duodenum. Henley has called this operation gastrectomy with replacement and has reported its performance in 110 patients, with 97% of the patients exhibiting a gain in weight after operation.*

Experimental evaluation of certain aspects of the nutritional status following this procedure of subtotal gastrectomy with jejunal replacement was performed.

METHOD OF STUDY

Eight healthy mongrel dogs were successfully subjected to subtotal gastrectomy with jejunal replacement. The operative procedure is illustrated in Figure 1. Seventy-five to 80% of the stomach was resected in each instance, and the Hofmeister method of removing the lesser curvature portion of the residual gastric pouch was used. A segment of proximal jejunum approximately 8 to 10 cm. long was isolated, the proximal end of the jejunal graft was closed, and the stomach was anastomosed to the side of the jejunal segment near the closed end. The distal end of the jejunal segment was anastomosed end-to-end to the duodenum. The interrupted small bowel was reunited by end-to-end anastomosis.

Two to three months after operation metabolism (intake-excretion) studies were performed. Four individual seven-day metabolism periods were conducted for each animal with the exception of one animal which had only two periods. Thus, there were a total of 30 seven-day metabolism periods in the experimental group subjected to subtotal gastrectomy with jejunal replacement. The animals were given constant daily diets calculated and chemically analyzed to furnish 80 calories per kilogram of body weight and containing 4 gm. of fat and 5 gm. of protein per kilogram of body weight. Any food not eaten was weighed and subtracted from the total intake. Fecal collection periods were demarcated with carmine markers. The feces for each period were weighed and homogenized in a Waring Blendor, and fat and total nitrogen were determined on aliquot samples. Nitrogen was determined with the use of Hoffman's photelometric modification of the Koch and McMeekin direct nesslerization Kjeldahl micromethod.[2] Fecal fat was determined by the Soxhlet method.

At the conclusion of the metabolism studies, the animals were placed on the normal animal hospital diet but were supplied excessive amounts and permitted to eat as much as they wished. Gastric motility was studied by feeding a barium-coated test meal. Serial roentgenograms were taken immediately after feeding the barium meal and at one-half-hour intervals for two hours. Animals were killed at intervals from 6 to 11 months after operation, with the exception of one animal which was inadvertently killed four months after operation. At autopsy the residual gastric pouch, jejunal segment, and duodenum were removed and the capacity (volume) of the remaining stomach and jejunal segment was determined.

Comparison was made with similar studies, previously performed, on 14 normal dogs (38 seven-day metabolism periods), 10 dogs subjected

From the Department of Surgery, University of Illinois College of Medicine.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 3, 1954.

This investigation was supported by a research grant (G-3628) from the National Institutes of Health, Public Health Service.

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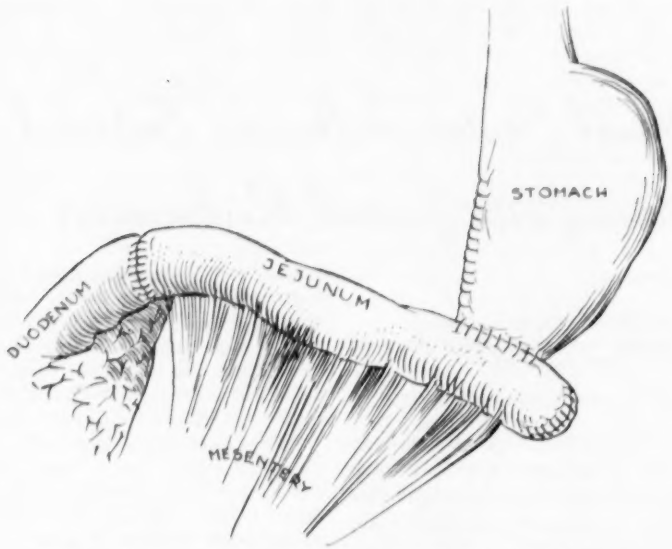

Fig. 1.—Diagram of subtotal gastrectomy with jejunal replacement.

to Billroth II type of subtotal gastrectomy (40 seven-day metabolism periods), and 10 dogs subjected to Billroth I type of subtotal gastrectomy (40 seven-day metabolism periods).[3]

RESULTS

Protein (Nitrogen) Assimilation

Graphic representation of the percentage of ingested nitrogen excreted in the feces during each seven-day metabolism study in the eight dogs subjected to subtotal gastrectomy with jejunal replacement is presented in Figure 2. The average percentage fecal nitrogen excretion was 11.9, with a range of 4.1% to 17.8%.

Fat Assimilation

Graphic representation of the percentage of ingested fat excreted in the feces during each seven-day metabolism period in the eight dogs subjected to subtotal gastrectomy with

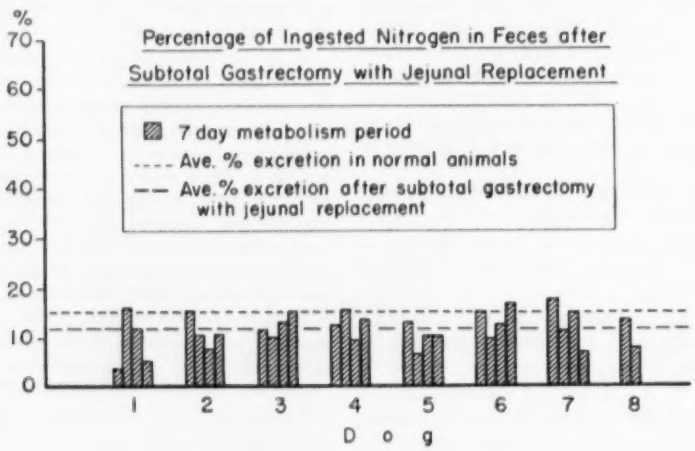

Figure 2

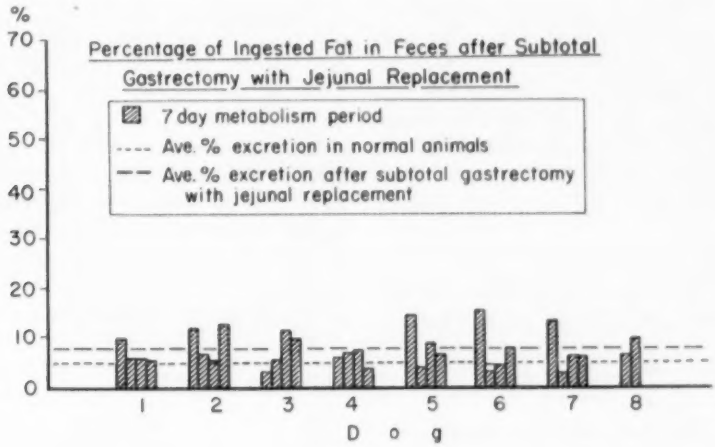

Figure 3

jejunal replacement is presented in Figure 3. The average percentage fecal fat loss was 7.8, with a range of 2.9% to 16%.

Comparison of Protein (Nitrogen) and Fat Assimilation

Graphic comparison of the average percentage fecal nitrogen and fat loss in normal animals and in animals after subtotal gastrectomy with jejunal replacement and Billroth I and Billroth II types of subtotal gastrectomy is presented in Figure 4. The average percentage fecal nitrogen loss following subtotal gastrectomy with jejunal replacement (11.9%) was not significantly different from the average percentage excretion noted in normal dogs (14.9%). The average percentage fecal nitrogen loss following Billroth I type of subtotal gastrectomy was greater than normal, but the difference was relatively slight. In contrast, the average fecal nitrogen loss following the Billroth II type of subtotal gastrectomy (24.4%) was appreciably greater than the average percentage loss noted in normal animals.

The average percentage fecal fat loss after subtotal gastrectomy with jejunal replacement (7.8%) was not significantly different from that noted in normal animals (4.9%). The average percentage fecal fat loss after

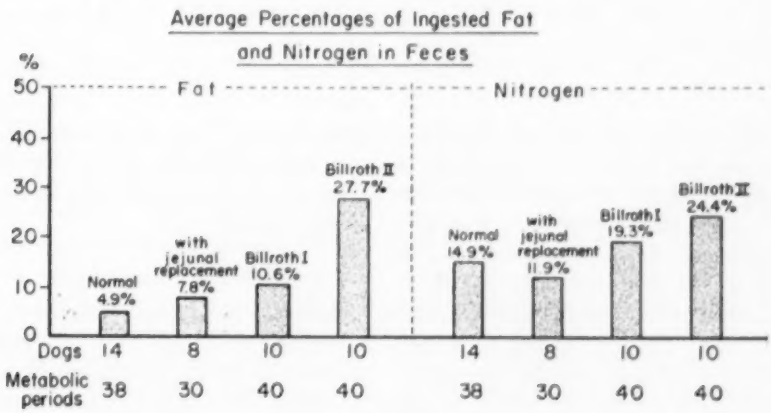

Figure 4

Billroth I type of subtotal gastrectomy (10.6%) was slightly greater than the fecal fat loss after subtotal gastrectomy with jejunal replacement. The difference was not, however, statistically significant. The average percentage fecal fat loss after Billroth II type of subtotal gastrectomy (27.7%) was markedly greater than in the other experimental groups.

Weight Study

Graphic representation of weight changes in the animals subjected to subtotal gastrectomy with jejunal replacement, Billroth I gastrectomy, and Billroth II gastrectomy is presented in Figure 5. Four of the eight jejunal replacement animals gained weight; two dogs showed no weight change, and two dogs lost weight. Six of the eight Billroth I dogs for which weight records were available gained wieght; one dog showed no weight change, and one dog lost weight. Five of the eight Billroth II animals for which weight records were available lost weight; two dogs gained weight, and one dog showed no weight change. All dogs were offered excessive amounts of food and permitted to eat as much as they wished. Thus, on ad libitum diets the majority of animals subjected to subtotal gastrectomy with jejunal replacement or Billroth I type of subtotal gastrectomy gained weight or maintained their weight, while the majority of animals subjected to the Billroth II type of gastrectomy lost weight. There appeared to be little difference in weight changes between the group of animals subjected to subtotal gastrectomy with jejunal replacement and the group of animals subjected to the Billroth I type of subtotal gastrectomy.

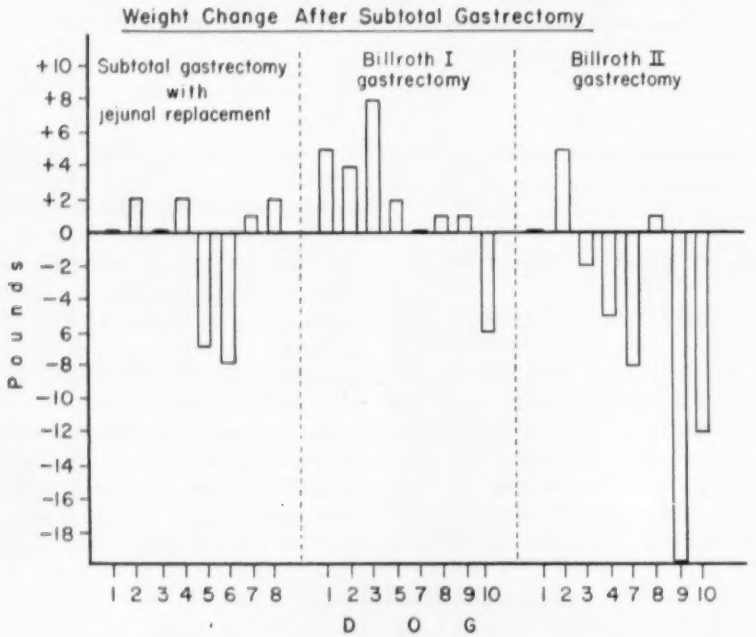

Figure 5

Gastric Emptying Time

Gastric emptying was compared by feeding a barium-coated test meal and noting the progress of the meal through the stomach by means of roentgenograms immediately after ingestion of the meal and at one-half-

hour intervals for two hours thereafter. Serial tracings were made of the barium outline of the stomach pouch, and the surface area of these tracings was measured by the use of the polar planimeter. The results of these studies are summarized in the Table. The average percentage of barium remaining in the stomach after each interval of time was essentially the same after subtotal gastrectomy with jejunal replacement and after Billroth I type of subtotal gastrectomy. However, the residual gastric pouch appeared to empty slightly more rapidly after the Billroth II type of gastrectomy.

Gastric Pouch Size

The capacities (volumes) of the individual residual stomachs of the experimental animals were measured at the time of autopsy.

Gastric Motility after Subtotal Gastrectomy

Type of Subtotal Gastrectomy	No. of Dogs	Percentage of Barium Remaining in Stomach			
		½ Hr.	1 Hr.	1½ Hr.	2 Hr.
With jejunal replacement	10	74.6	52.8	33.4	16.7
Billroth I	9	70.0	50.2	30.3	13.6
Billroth II	8	60.4	39.3	20.6	5.1

The average residual stomach capacity of eight dogs subjected to subtotal gastrectomy with jejunal replacement was 65 cc. (range of 35 to 100 cc.) plus an average jejunal segment capacity of 34 cc. (range 22 to 56 cc.), or an average combined capacity of 99 cc. (range 57 to 145 cc.), as compared with an average residual stomach capacity of 55 cc. (range of 37 to 135 cc.) in eight dogs subjected to the Billroth I type of subtotal gastrectomy and an average residual capacity of 118 cc. (range 37 to 230 cc.) in eight dogs subjected to Billroth II type of subtotal gastrectomy. Thus, the more normal assimilation of fat and protein after subtotal gastrectomy with jejunal replacement and Billroth I type of gastrectomy as compared with the assimilation after Billroth II type of gastrectomy cannot be attributed to greater residual gastric pouch capacity.

COMMENT

Fat assimilation, protein assimilation, and weight status after subtotal gastrectomy with jejunal replacement and after Billroth I type of gastrectomy were experimentally found to be markedly better than after the Billroth II type of gastrectomy. The nutritional differences experimentally noted between subtotal gastrectomy with jejunal replacement and the Billroth I type of subtotal gastrectomy were so slight as to suggest that the main if not the only function of the interposed jejunal segment was to serve as a conduit to connect the stomach and the duodenum. The jejunal segments did not retard gastric emptying into the duodenum in this experimental series. Thus the jejunal segments exhibited little or no effective reservoir function. Goligher and Riley [4] have previously reported clinical observations of lack of satisfactory reservoir function of colon segments interposed between the residual stomach and duodenum. Accordingly, as Harkins and his co-workers [5] have aptly demonstrated that it is possible to do a high (75%) gastric resection for the ulcer diathesis and perform a Billroth I type of anastomosis in a large percentage of cases, there would seem to be but limited indication for the added procedure of jejunal replacement.

CONCLUSIONS

Nutritional status was studied in eight experimental dogs after subtotal gastrectomy with jejunal replacement and was compared with the nutritional status in normal dogs and in dogs after Billroth I and Billroth II types of subtotal gastrectomy.

The average percentage fecal nitrogen loss after subtotal gastrectomy with jejunal replacement (11.9%) was not significantly different from the average percentage excretion noted in normal dogs (14.9%). The average percentage fecal nitrogen loss after Billroth I type of subtotal gastrectomy was greater than normal, but the difference was relatively slight. The average fecal nitrogen loss after the Billroth II type of subtotal

gastrectomy (24.4%) was appreciably greater than the average percentage loss noted in normal animals.

The average percentage fecal fat loss after subtotal gastrectomy with jejunal replacement (7.8%) was not significantly different from that noted in normal animals (4.9%). The average percentage fecal fat loss following Billroth I type of subtotal gastrectomy (10.6%) was slightly greater than the fecal fat loss after subtotal gastrectomy with jejunal replacement. The difference was not statistically significant, however. The average percentage fecal fat loss after Billroth II type of subtotal gastrectomy (27.7%) was markedly greater than the other experimental groups.

On ad libitum diets the majority of animals subjected to subtotal gastrectomy with jejunal replacement or to Billroth I type of subtotal gastrectomy gained weight or maintained their weight, while the majority of animals subjected to the Billroth II type of gastrectomy lost weight.

The jejunal segments did not retard gastric emptying into the duodenum in this experimental series.

The slight nutritional differences experimentally noted between subtotal gastrectomy with jejunal replacement and the Billroth I type of subtotal gastrectomy suggest that the principal function of the interposed jejunal segment is to serve merely as a conduit to connect the stomach and the duodenum.

The data obtained in this study suggest that the procedure of subtotal gastrectomy with jejunal replacement has no appreciable nutritional advantage over direct anastomosis of the residual stomach to the duodenum by the Billroth I technique.

DISCUSSION

DR. JOHN T. REYNOLDS, Chicago: I would like to suggest that Dr. Henley's procedure does have a very practical application for those patients who have had a fairly radical gastrectomy, who have a serious dumping syndrome.

Although we have done eight or nine of the Henley procedures, six were elective and two were done for what we thought was very definite dumping syndrome. These two were done on patients who, although they had had absolutely satisfactory results as far as complete and total relief of their ulcer syndromes was concerned, had marked weight loss and all of the other associated symptoms of dumping syndrome. However, the weight loss was the most significant factor.

These patients could not work; they had no strength, and both of these men had been big, husky men, weighing 180 lb. and 190 lb. respectively. When they came back to the hospital for the Henley procedure they weighed around 120 lb. Both patients complained of repeated and almost constant vomiting of bile, even during the interval when they were not being fed.

The Henley loop, the isolated loop of jejunum, was so rotated that the oral portion of the jejunal segment was anastomosed to the stomach and the aboral to the duodenum. A vagotomy was done in both of these patients.

The first patient called me on the telephone from his bed 24 hours after being operated upon and asked, "Why didn't you do this operation the first time?" For the first time since his operation he had no bile in his stomach. Since then he has gained 30 lb. in weight and is quite strong.

The second man was not quite as positive in his recognition of his benefit, but he has now gone back to work.

This operation has a very important place in relieving the patients who have small stomach syndrome after the extensive and high gastric resection.

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Relationship of Corticotropin (ACTH) to the Healing of Gastrointestinal Anastomoses

Experimental and Clinical Observations

THOMAS GEOGHEGAN, M.D.
and
BROCK E. BRUSH, M.D., Detroit

Several surgeons have noted that wounds in patients under treatment with corticotropin (ACTH) or cortisone heal without delay, although early experimenters * demonstrated that these agents inhibit healing. For example, Cole and his associates[3] used corticotropin and cortisone in a variety of surgical conditions without encountering difficulties with wound healing. Brush, Monto, and coworkers [4] treated 13 patients with thrombocytopenic purpura with corticotropin or cortisone before and after splenectomy and had no wound complications. Szilagyi and associates [5] reported similar results in hyperthyroid patients prepared for thyroidectomy with corticotropin.

The warnings of Hume and Moore [6] and of Zimmerman [7] against the use of corticotropin and cortisone in the presence of anastomoses in the gastrointestinal tract carry considerable weight when the consequences of anastomotic breakdown are contemplated. In an effort to evaluate this problem we have reviewed the outcome of corticotropin and cortisone administration in patients in this hospital with gastrointestinal anastomoses. The clinical data compared with information obtained from a controlled study of intestinal anastomoses in corticotropin-treated dogs form the basis of this report.

Department of General Surgery, Henry Ford Hospital.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 3, 1954.

* References 1 and 2.

CLINICAL OBSERVATIONS

Corticotropin and cortisone were administered to nine patients in the immediate postoperative period following gastrointestinal anastomoses between 1951 and 1953. In each instance malnutrition or poor general condition of the patient was the reason for use of the hormone. Six patients underwent subtotal gastric resection. In this group two patients received such small doses of corticotropin during such a short period of time that a clinical effect from the medication was improbable. The remaining four were given from 0.25 to 4.0 units of corticotropin per kilogram daily for from 6 to 15 days postoperatively (Fig. 1). Two of these patients, who were operated upon for peptic ulcer, had gastroduodenal anastomoses. The others, one with gastric carcinoma and one with chronic duodenal ulcer, had gastrojejunostomies. These patients responded to corticotropin as judged by decrease in their circulating eosinophiles. All of the patients in this group left the hospital after the usual interval following procedures of this nature. Peritonitis and fistula formation indicating leakage at the site of anastomosis did not occur.

Two additional patients, on whom extensive procedures were carried out, died in the postoperative period while on corticotropin therapy (Fig. 2).

Case 1.—W. K. (633194), a 38-year-old white man, was admitted to the hospital with symptoms resembling peptic ulcer. He had lost 20 lb. during the preceding year. He was operated upon by Dr. D. Emerick Szilagyi, who found a diffusely infiltrating carcinoma of the stomach with node involvement but no evidence of liver metastases. A total gastrectomy with resection of the spleen and the tail of the pancreas was done, and the ileocecal segment was transposed to the region of the resected stomach. Anastomoses were made between the ileum and esophagus and between the cecum

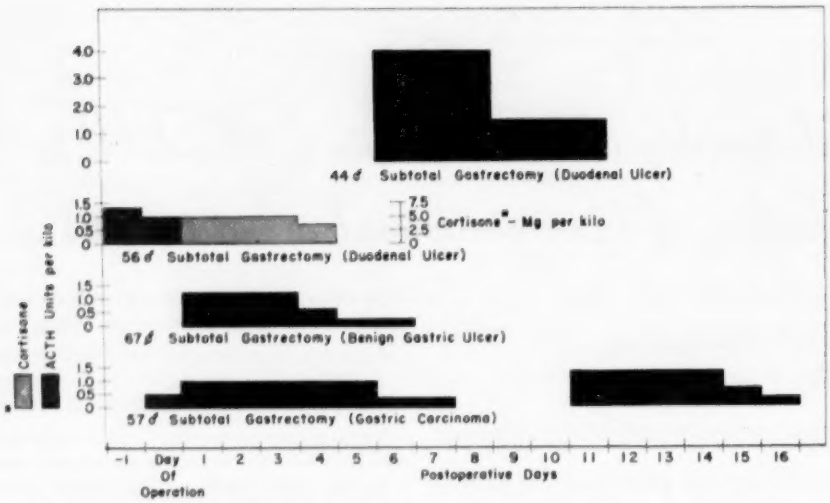
44 ♂ Subtotal Gastrectomy (Duodenal Ulcer)
56 ♂ Subtotal Gastrectomy (Duodenal Ulcer)
67 ♂ Subtotal Gastrectomy (Benign Gastric Ulcer)
57 ♂ Subtotal Gastrectomy (Gastric Carcinoma)
Cortisone* - Mg per kilo
Cortisone
ACTH Units per kilo
Day Of Operation
Postoperative Days

Fig. 1.—Dosage levels of corticotropin and cortisone in four patients after gastrectomy.

and the duodenum. The ends of the proximal ileum and the distal ascending colon were joined. On the day of operation and for the first five postoperative days the patient was given corticotropin in doses of 0.25 to 1.25 units per kilogram daily in addition to penicillin and streptomycin. The patient's progress was good and he began taking oral fluid on the fifth postoperative day. No corticotropin was administered from the sixth to the eighth postoperative day. Because of a gradual decline in the patient's condition, corticotropin was again given on the 9th day and continued until the 12th day when the patient died with symptoms and signs resembling those of peritonitis.

Postmortem examination showed the cause of death to be a severe hemorrhagic pancreatitis with mesenteric fat necrosis and peritonitis. The anastomoses were, however, all intact. Microscopic examination of the suture lines showed good healing with considerable fibroblastic proliferation and

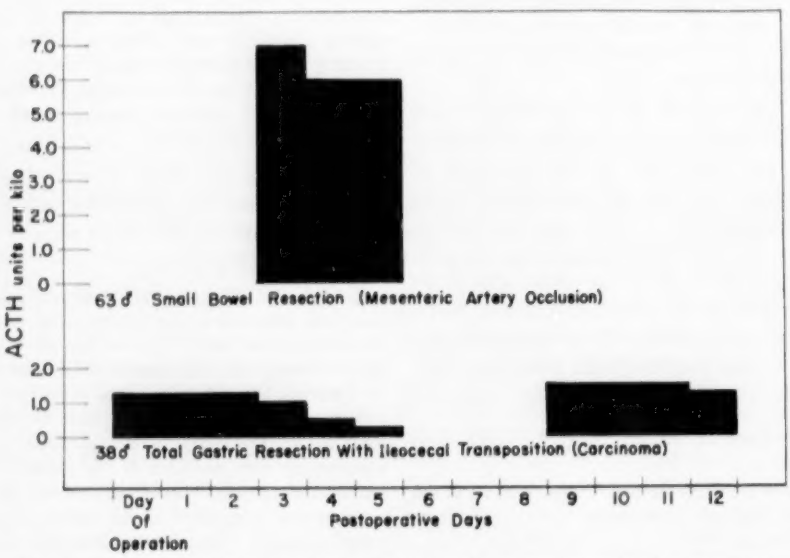
63 ♂ Small Bowel Resection (Mesenteric Artery Occlusion)
38 ♂ Total Gastric Resection With Ileocecal Transposition (Carcinoma)
ACTH units per kilo
Day Of Operation
Postoperative Days

Fig. 2.—Dosage levels in two patients who died while on corticotropin after operation.

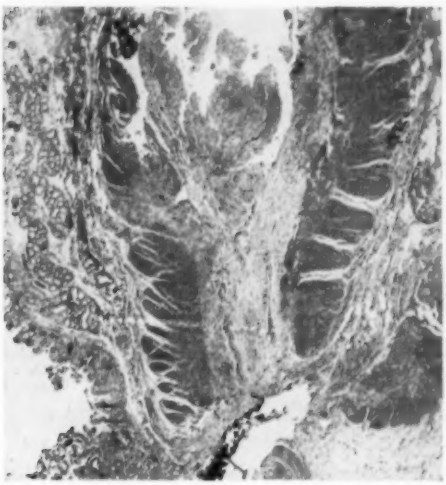

Fig. 3.—Section of anastomosis between duodenum and cecum from patient who died while on corticotropin therapy after gastric resection and ileocecal transposition. Inverted duodenum can be seen on the left, cecum on the right. A nonabsorbable (silk) suture is in the lower portion of the section. Hematoxylin and eosin, reduced to 4/7 of mag. × 30.

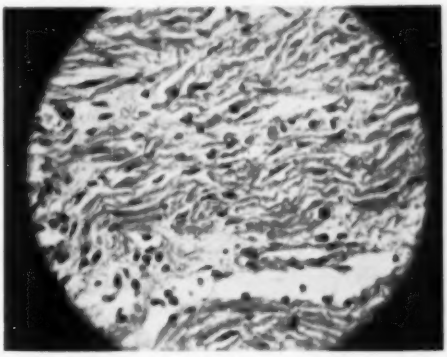

Fig. 4.—Section from the line of union of anastomosis shown in Figure 3 demonstrating proliferation of fibroblasts and vascular ingrowth. Hematoxylin and eosin, reduced to 1⅝₃ of mag. × 430.

vascularity. Sections from the anastomosis between the colon and duodenum in this case are shown in Figures 3 and 4.

The second patient who died while on corticotropin treatment had a resection of gangrenous jejunum, ileum, and ascending colon caused by mesenteric thrombosis. A myocardial infarction resulted in the patient's death on the sixth post-

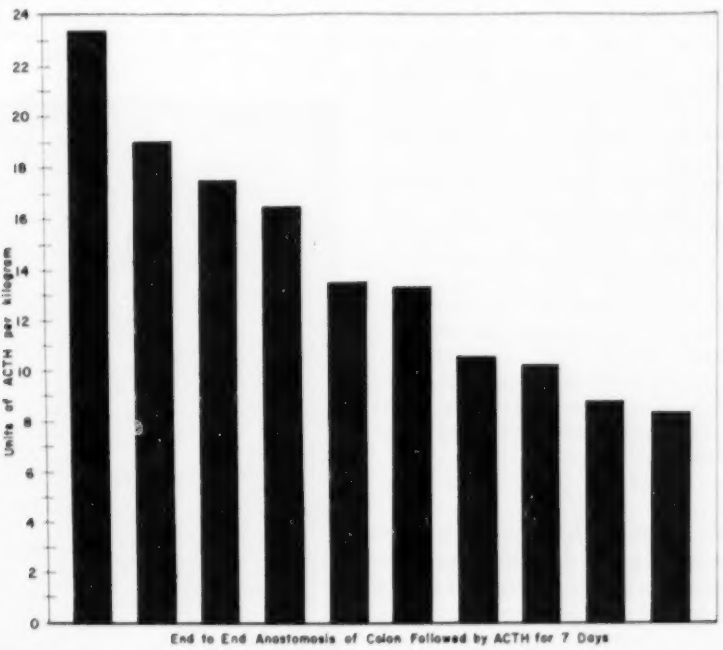

Fig. 5.—Bar graph of corticotropin dosage levels in 10 dogs after anastomoses of the colon. Each bar represents the daily dose of corticotropin for an individual animal, begun on the day of operation and continued for seven days postoperatively.

operative day. At autopsy the anastomosis between the colon and jejunum showed good healing with no evidence of leakage. This patient received daily doses of 6.0 to 7.0 units of corticotropin per kilogram between the third and sixth postoperative day as shown in Figure 2.

EXPERIMENTAL STUDY

In the preceding cases the lack of evidence of corticotropin inhibition of anastomotic healing might be attributed to the small size of the dosage in each instance. In order to determine whether a demonstrable inhibitory effect could be obtained with massive doses, dogs were given corticotropin after end-to-end anastomoses of the colon. The hormone was administered in amounts much larger than those used clinically, the largest being equivalent to over 1600 units per day in an adult male patient (Fig. 5).

METHOD

Twenty full-grown dogs ranging in weight from 6.4 to 18.9 kg. were subjected to end-to-end anastomosis of the colon by the Parker-Kerr basting stitch method, after the transverse colon was divided between clamps without interference with its blood supply. A single layer of nonabsorbable (silk) sutures was used to restore union. Ten of the dogs were given daily subcutaneous injections of repository corticotropin upon closure of the abdomen and for seven days postoperatively. Each animal was given 160 units per dose, so that the dosage ranged from 6.4 to 23.4 units per kilogram per day depending upon the weight of the animal. The 10 control dogs were given no medication. All dogs were maintained on milk postoperatively. No antibiotics were used. On the seventh postoperative day each animal was killed. The bursting strength of the anastomoses was determined by introducing compressed air into the lumen of the submerged bowel to the point at which bubbles of air escaped from the suture line. Sections of the anastomoses were stained with hematoxylin and eosin for microscopic examination.

RESULTS

Both the control and treated animals remained in good health following operation. Daily eosinophile counts of the treated dogs showed a total or near-total eosinopenia while these of the control dogs dropped 10 to 50% from preoperative levels. No suture line leakage occurred in the treated or control series. The results of the bursting strength determinations in the treated and non-treated animals are shown in the Table. Each series

Bursting Strength of Colon Anastomoses in Dogs with and without Corticotropin

Treated Dogs			Nontreated Dogs	
Dog No.	Corticotropin Units per Kilogram per Day	Anastomotic Bursting Strength, Mm. Hg	Dog No.	Anastomotic Bursting Strength, Mm. Hg
1	8.4	230	11	196
2	9.3	210	12	280
3	10.3	140	13	120
4	10.6	150	14	200
5	13.4	160	15	300
6	13.5	340	16	210
7	16.4	260	17	365
8	17.5	225	18	345
9	19.0	230	19	325
10	23.4	300	20	230
Mean		224		257
"t" value		10.98		11.4
Probability		0.001		0.001
"t" value of differences		1.08		

Fig. 6.—Section of end-to-end anastomosis of the colon in dog given 16.4 units of corticotropin per kilogram per day. Compare with Figure 7. Hematoxylin and eosin; reduced to ½ of mag. × 40.

Fig. 7.—Section of anastomosis of colon in non-treated control dog. Hematoxylin and eosin, reduced to ⅙ of mag. × 40.

had a statistically significant mean bursting strength value. There was no significant difference between the treated and non-treated groups. A comparison of the sections revealed a similar proliferation of fibroblasts, vascular response, and reaction around the nonabsorbable (silk) suture material at the anastomoses in the treated and control animals (Figs. 6 and 7).

COMMENT

Because of the apparent lack of inhibitory action by corticotropin on the anastomoses in the patients in this series, the matter of hormone dosage used in the experimental study is important. By employing graded doses beginning at a higher level than the largest clinical amount per kilogram of body weight, it might be expected that a level could be found above which disruption of the anastomosis and peritonitis would occur. This was not the case. In fact, there was no evident decrease in the strength of the anastomoses with increase in the amounts of corticotropin administered (Table). The corticotropin preparation was a repository suspension prepared to exert hormonal stimulation of the adrenal cortex over a prolonged time interval.

These observations are in agreement with results of other surgical experiments dealing with the effect of cortisone on the healing of per primam wounds. Taylor and coworkers [8] in experiments on skin wounds in rats and Cole and others [9] in experiments on skin wounds in dogs were unable to produce retarded wound healing with cortisone. Muller and his associates [10] obtained similar results in a study of the effect of cortisone on vascular anastomoses in dogs.

Although much of the early enthusiasm for the clinical use of corticotropin and cortisone in a number of surgical conditions has not withstood the test of time, there are certain situations in which these agents are valuable. The accumulation of clinical and laboratory data from a number of centers seems to indicate that corticotropin and cortisone may be given to surgical patients without adverse effect on wounds which would heal well otherwise.

SUMMARY AND CONCLUSIONS

In a small series of patients treated with corticotropin (ACTH) and cortisone after gastrointestinal anastomoses treatment with corticotropin and cortisone did not cause leakage at the suture lines. Postmortem examination of the anastomoses in two patients showed satisfactory healing. In experimental colon anastomoses in dogs corticotropin in extremely high doses did not alter the strength or histological characteristics of the anastomoses as compared with non-treated controls.

DISCUSSION

DR. BROCK E. BRUSH, Detroit: I just want to point out again two important points about patients who are receiving corticotropin or cortisone.

The first one is the fact that these hormones have a great effect on the acid and pepsin secretion of the stomach. There have been several reports of reactivation or perforation of an ulcer when a patient was on cortisone therapy.

The other point that we must know when a patient comes to surgery is whether or not he has been receiving cortisone for any reason. We know that exogenous cortisone will cause a physiological atrophy of the patients' adrenals and when the exogenous supply is discontinued the patient may be for several weeks or even months in a state approaching Addison's disease. An operation or even an anesthetic in this critical period may be

fatal unless additional cortisone is administered. A test of adrenal function should always be done if the patient has had any cortisone in the previous three to four months.

It is just as important to determine whether a patient has been on cortisone as it is to determine whether he uses insulin.

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Familial Occurrence of Congenital Hypertrophic Pyloric Stenosis

Report of Cases in Siblings and a Cousin and Review of the Literature

ANGUS L. CAMERON, M.D., Minot, N. D.

While congenital hypertrophic pyloric stenosis occurs quite regularly and not at all infrequently, it apparently afflicts more than one member of a family very rarely and familial occurrences of this kind are distinctly sporadic. In this country there are now approximately 4,000,000 live births annually; so, at the rate of 3 pyloric stenosis cases per 1,000, about 12,000 infants are found to have the disease every year. But, according to case reports, American clinicians do not observe its familial occurrence once a year. In fact, as we shall see, there are recorded in the world literature for all time fewer than 125 authentic cases, and if all reports which have been published were counted, irrespective of their validity, the number would still be extremely small.

For this reason alone, then, all familial occurrences should be reported, but for another and perhaps better one they merit publication, and that is to provide medical geneticists material for their investigations into the possible hereditary nature of congenital pyloric stenosis and also its etiology. These investigators are wholly dependent upon clinicians for reliable case reports. As Bauer [1] pointed out a number of years ago, the latter can "depend on the cooperation of the geneticist and the statistician to perform the less esteemed labor of statistical analysis" once they have the case reports.

From the Department of Surgery, Northwest Clinic.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Dec. 3, 1954.

These records, of course, should concern only verified instances of familial occurrences of the disease and reveal also sufficient data for the work at hand. Unfortunately, many are deficient in both respects. To often an author has failed to establish the validity of his claim by neglecting to prove that pyloric stenosis actually occurred in any or all of the family group. So-called medical diagnoses, of which there are many, are no doubt more reliable now than they were years ago, but still they are too fallible for undoubted assurance of the presence of a typical pyloric deformity. The latter must be seen at operation or autopsy to provide satisfactory proof that it exists. Anything short of this should not be referred to as valid, bona fide, verified, or authentic and is not in this paper. Strict observance of this rule when the literature is searched for case reports will, no doubt, result in the discarding of some which are actually valid. But for one who is striving to be factual there is no alternative to scrutinizing the records with care to eliminate all cases which are at all doubtful. When this is done, some claims, noteworthy for the number of familial occurrences reported, will have to be refused acceptance. Since 1947 three have appeared in this country in which the number of cases is relatively large in each instance, but among them only the claims of Nafe [2] have been found to be verified. He observed 6 instances of 2 affected members in one family among 129 infants treated surgically for pyloric stenosis. But, on the other hand, Schaefer and Erbes [3] have left the reader in doubt about all but one of the nine cases their re-

port contains, and Grimes, Bell, and Olney's [4] paper fails to provide verifying evidence for any of the 14 familial occurrences mentioned there.

Brendle's [5] often quoted report of four afflicted families, none of whom he saw when sickness was present, is another one which is found to be completely unsatisfactory upon scrutiny. He failed to prove his claim in a single case. An example of some of the evidence which he cited is as follows: "The children's grandmother told us that one of her sons, the brother of the children's father, had suffered from pylorospasmus." Without doubt, the recollections of relatives occasionally merit serious consideration but should never be accepted for more than they are worth.

TABLE 1.—*Familial Occurrences of Congenital Pyloric Stenosis Observed by the Author* *

	Number	Birth Dates
1. Madsen siblings	2	1940 and 1943
2. Stephan siblings	3	1950, 1951 and 1953
3. Sandback siblings	2	1948 and 1954
4. Savchenko and Peterson first cousins	2	1954 and 1953

* Besides these nine infants who are known to have had pyloric stenosis, two more very likely did. They are the oldest of the Madsen siblings and their first cousin.

A glaring violation of this rule is cited here to emphasize how unreliable some secondhand reports can be. According to Cockayne and Penrose,[6] Cautley and Dent [7] reported an instance of congenital pyloric stenosis in a great-aunt and her nephew. Now what they did say in 1902, when reporting a successful pyloroplasty on the nephew, who was found to have the disease, was that "it is worth recording, though we lay no stress on what may be merely a coincidence, that the grandmother on the father's side lost a sister in infancy whose death was ascribed to nothing passing through the stomach.

From all that has just been said, it is apparent that the reviewer of the literature on this subject, if he is intent upon accuracy, has a labor to perform like that of the geneticist and the statistician, which is less esteemed than some other kinds.

The material upon which this paper is based is found in Tables 1 to 9. In Table 1 are listed four familial occurrences of congenital hypertrophic pyloric stenosis which I have observed at the Northwest Clinic, Minot, N. D., during the last 17 years. Sixty-four families with 68 affected infants have come under our observation during this time. All the infants had a typical pyloric deformity, and all were treated surgically for it by means of the Fredet-Ramstedt technique. Strange to say, two of these four family occurrences of the disease were observed within the last few weeks, during the month of November, 1954.

These cases are reported here in sufficient detail, it is hoped, to meet the requirements of the medical geneticists fairly satisfactorily. In addition to this, as already intimated, the literature on the subject has been reviewed rather extensively. Tables 2 to 8, inclusive, contain the listings in chronological order of the several categories of relationship which have been found and are considered verified case reports. In Table 9 are the unverified reports together with a notation in the legend for each one appearing there. This attempt to weed out doubtful claims of familial occurrence of the disease and also those totally without verification would seem to be as timely as it is without precedent.

CASE REPORTS

1. THE PALMER MADSEN FAMILY (Fig. 1)

Only two of the five Palmer Madsen children were seen at the Northwest Clinic, namely, Eleanor and Melvin, who were the second- and fourth-born, respectively, and who were both treated surgically for pyloric stenosis. Their older brother, Norman, the first-born of the five siblings, became a severe feeding problem shortly after birth, according to the parents, and so remained for several months. He suffered from projectile vomiting and became greatly emaciated. He was born at home on a farm on Oct. 15, 1938, and was under the care there for some time of the family doctor who delivered him. Later, another doctor in a nearby town took over his medical care but with results which were unsatisfactory to the parents. They then took things into their own hands, relying mainly upon a Horlick's malted milk formula. Recovery finally occurred, and the child is living and well today.

TABLE 2.—*Familial Occurrence in Two Siblings Born Singly* *

	Author	Nationality of Author	Date of Report	Sex	Birth Rank
1.	Ramstedt [16]	German	1912	Male	3d, 4th
2.	Hammond [17]	American	1913	Male, female	.....
3.	Kerley [18]	American	1918	.....	1st, 2d
4.	Heekes [19]	British	1926	Male	1st, 2d
5.	Bratusch-Marrain [36]	Austrian	1928	One male, one unknown	Successively born
6.	Cockayne, Monerieff, and Sheldon [37]	British	1928	Male	2d and 3d of 5 siblings
7.	Hall [38]	American	1929	Male	4th and 5th
8.	Higgins †	American	1929	.....	Successively born
9.	Tribble [39]	American	1933	.....	1st and 3d
10.	Lario ‡	American	1935	Male	.....
11.	Thomson and Gaisford [41]	British	1935	Female	1st and 2d
12.	Orgeolet [42]	French	1936	Male, female	Successively born
13.	Orgeolet [42]	French	1936	Male	.....
14.	Orgeolet [42]	French	1936	Male, female	.....
15.	Orgeolet [42]	French	1936	Male	.....
16.	Halbertsma [6]	Dutch	1936	Male, female	.....
17.	Halbertsma [6]	Dutch	1936	Male	.....
18.	Halbertsma [6]	Dutch	1936	Male, female	.....
19.	Jelks [43]	American	1937	Female	1st and 3d of 5 siblings
20.	Romano and McFetridge [44]	American	1938	Male, female	3d, 4th
21.	Heems §	French	1938	.....	.....
22.	Lereboullet [45]	French	1938	Male	.....
23.	Lereboullet [45]	French	1938	One known male	.....
24.	Blacher [47]	German	1939	Male	1st, 5th
25.	O'Donnell and Klein [48]	American	1941	Male	.....
26.	Cranmer [49]	American	1941	Female	.....
27.	Marquezy [50]	French	1944	Male	1st, 3d
28.	Collins [51]	American	1946	.....	Unknown among 10 siblings
29.	Kaufman [53]	American	1947	Male	1st, 2d among 3 siblings
30-32.	Akin and Forbes [55]	American	1947	.....	.....
33.	Schachter [54]	French	1948	Male	.....
34.	Planson and Chavelet [55]	French	1949	Female	4th and 5th
35.	Ward-McQuaid, and Parritt [56]	British	1950	.....	.....
36.	LeGac [57]	French	1951	Male	.....
37.	Huang [58]	Chinese	1951	Male, female	1st, 2d
38-40.	Wood and Smellie [59]	British	1951	.....	.....
41-51.	McKeown, MacMahon, and Record [35]	British	1951	.....	.....
52.	Keizer [34]	French	1952	Male, female	1st, 2d
53.	Fenwick [60]	British	1953	Male	1st, 4th
54-59.	Nafe [3]	American	1954	.....	.....

* In one infant in each of the reports by Ramstedt and Bratusch-Marrian the diagnosis was verified at autopsy. In all the rest listed here the pylorus was seen at operation, with the possible exception of a case or so found in the reviews of Akin and Forbes. Two more such instances mentioned by Bilderback in 1928 are not included in this table because they lack verification. The brothers reported by Schachter and Planson had different mothers. In a personal communication, Nafe has just informed me that in one of the six instances of two affected members in one family reported in 1947 a third infant with pyloric stenosis has been recently operated upon by him, making three affected siblings in this one family.

† Higgins, in discussion on Hall.[38]

‡ In discussion on Strong.[40]

§ Cited by Ponyanne.[45]

At about the time of Norman's recovery a first cousin, Ellen Sheelar, was born on a nearby farm. This was on March 23, 1939. She was likewise the first-born of a family of siblings which finally numbered four girls and one boy. Her father was a brother of Norman's mother and her mother was a sister of Norman's father. She, too, soon became a serious feeding problem, suffering from projectile vomiting and extreme emaciation, according to Norman's mother, who told me the story in 1954. Ellen's care was in the hands of the family physician who delivered her and who was the second doctor to treat Norman. Ellen died in convulsions at the age of 2 months. No autopsy was performed. None of the other siblings in this family had any similar trouble, and all are living and well today.

The ages of the father and mother of the Madsen children when their first child, Norman, was born were 21 and 26 years, respectively, and the ages of the father and mother of the Sheelar children when their first child, Ellen, was born were 26 and 28 years, respectively. Case reports of the two Madsen children with known pyloric stenosis are given here.

CASE 1.—Eleanor, second child, was born in a maternity home at Velva, N. D., Feb. 21, 1940, under the care of the family physician, Dr. M. I. H. Kauffman. She became a feeding problem at the age of 3 weeks, when she began to have projectile

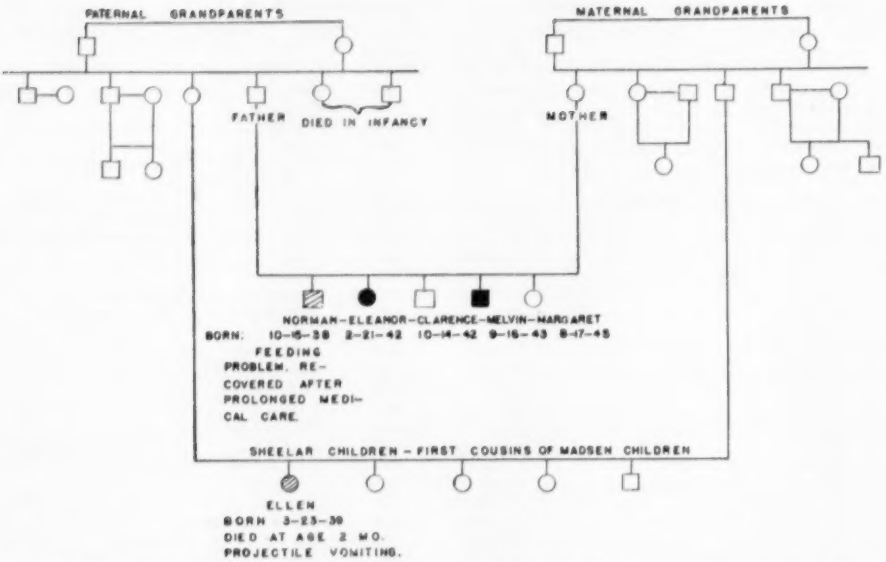

Fig. 1.—The Madsen family pedigrees. Eleanor and Melvin Madsen were treated surgically for congenital hypertrophic pyloric stenosis at Trinty Hospital, Minot, N. D. on April 17, 1940, and Oct. 14, 1943, respectively. Their older brother, Norman, probably had the same affliction but recovered without an operation. Their first cousin, Ellen Sheelar, likewise probably had pyloric stenosis. She died at the age of 2 months.

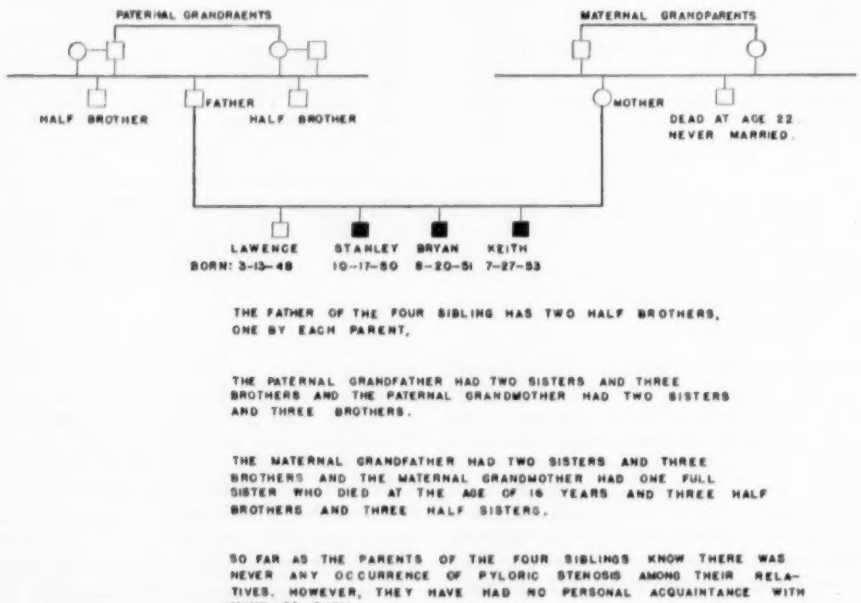
PATERNAL GRANDPARENTS
MATERNAL GRANDPARENTS
HALF BROTHER
FATHER
HALF BROTHER
MOTHER
DEAD AT AGE 22
NEVER MARRIED.
LAWENCE
BORN: 3-13-48
STANLEY
10-17-50
BRYAN
8-20-51
KEITH
7-27-53
THE FATHER OF THE FOUR SIBLING HAS TWO HALF BROTHERS, ONE BY EACH PARENT.
THE PATERNAL GRANDFATHER HAD TWO SISTERS AND THREE BROTHERS AND THE PATERNAL GRANDMOTHER HAD TWO SISTERS AND THREE BROTHERS.
THE MATERNAL GRANDFATHER HAD TWO SISTERS AND THREE BROTHERS AND THE MATERNAL GRANDMOTHER HAD ONE FULL SISTER WHO DIED AT THE AGE OF 16 YEARS AND THREE HALF BROTHERS AND THREE HALF SISTERS.
SO FAR AS THE PARENTS OF THE FOUR SIBLINGS KNOW THERE WAS NEVER ANY OCCURRENCE OF PYLORIC STENOSIS AMONG THEIR RELATIVES. HOWEVER, THEY HAVE HAD NO PERSONAL ACQUAINTANCE WITH MANY OF THEM.

Fig. 2.—The Stephan family pedigree. Stanley Stephan was treated surgically for congenital hypertrophic pyloric stenosis at the Good Samaritan Hospital, Williston, N. D. on Nov. 7, 1950, by W. A. Wright, M.D. His two brothers were both treated surgically for the same affliction at Trinty Hospital, Minot, N. D., Bryan on Sept. 25, 1951, and Keith on Aug. 11, 1953.

vomiting and to lose weight. Her birth weight was 8 lb., 2 oz., and when she was first seen at the Northwest Clinic, on April 15, 1940, it was 5 lb., 9 oz. She was admitted to Trinity Hospital on that date on the pediatric service of Dr. Ralph Dyson, who made a diagnosis of pyloric stenosis. Two days later, April 17, 1940, I performed a Fredet-Ramstedt operation on her using local procaine hydrochloride (Novocaine) anesthesia. A large olive-shaped pyloric deformity was found. Convalescence was uneventful except for infection of both ears, which required myringotomy on April 28 and 29. She was discharged from the hospital in good condition May 6, 1940.

CASE 2.—Melvin, fourth child, was born on Sept. 16, 1943, in the same maternity home and under the care of the same physician as was his sister, Eleanor. His birth weight was 8 lb., 5 oz. He began vomiting a few days after birth, at first only once a day, but after his 10th day of age the vomiting occurred after nearly every feeding and was projectile. Weight loss soon became severe. He was first seen at the Northwest Clinic by Dr. Ralph Dyson, Oct. 12, 1943, and was admitted to Trinity Hospital the same day with a diagnosis of pyloric stenosis. Two days later, Oct. 14, 1943, I performed a Fredet-Ramstedt operation with the patient under local procaine hydrochloride anesthesia; a large olive-shaped pyloric deformity was found. Convalescence was uneventful. He was discharged from the hospital in good condition Oct. 26, 1943.

As Figure 1 shows, a fifth and last child, Margaret, was born to the Madsens on Aug. 17, 1945. Like her brother Clarence, born Oct. 14, 1942, she had no symptoms of pyloric stenosis.

In conclusion of the story, then, of the Madsen and Sheelar families, it is certain that Eleanor and Melvin Madsen had pyloric stenosis and their brother Norman and double cousin, Ellen Sheelar, may very well have had the same affliction.

2. THE CHARLES STEPHAN FAMILY (Fig. 2)

The first child, Lawrence, was born March 13, 1948, in the Good Samaritan Hospital, Williston, N. D. The father's and mother's ages at this time were 23 and 25 years, respectively. Lawrence had no feeding difficulty and has remained well. But the story of his three brothers who were successively born in the same hospital is quite different. The four brothers constitute all the children in the family. They were born within a short period of time—a little longer than five years—from March 13, 1948, to July 27, 1953. The case reports of the three afflicted ones are as follows:

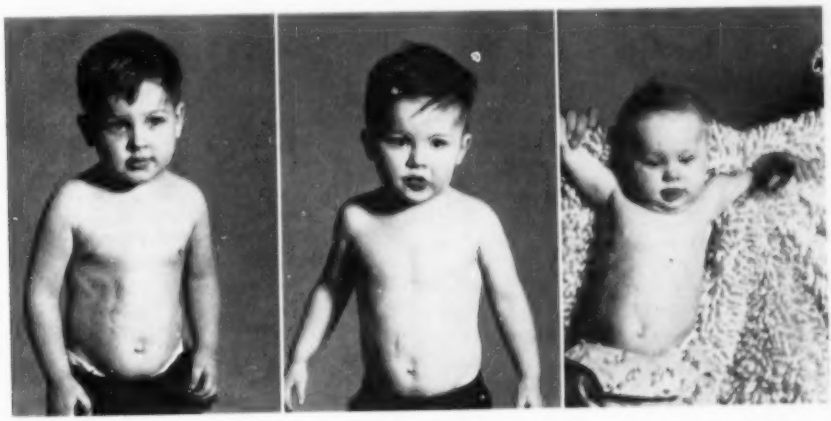

Fig. 2A.—The three Stephan brothers, Stanley, Bryan, and Keith, who had congenital hypertrophic pyloric stenosis.

CASE 1.—Stanley, born Oct. 17, 1950, began to vomit eight days later, according to Dr. C. M. Lund,* of Williston. The vomiting was projectile and was accompanied by severe weight loss. A diagnosis of pyloric stenosis was made and on Nov. 7, 1950, a Fredet-Ramstedt operation was performed by Dr. W. A. Wright, of Williston, a typical olive-shaped pyloric deformity being found. Recovery followed and the infant was discharged from the hospital Nov. 26, 1950.

CASE 2.—Bryan was born Aug. 20, 1951. His postnatal hospitalization was four days. At the age of two weeks, when separated from his parents for three days, he began to vomit. This symptom was mild for a few days and then increased in frequency and severity, finally becoming projectile and very aggravated. Weight loss occurred. Medical management gave no relief. The baby was first seen at

* Personal communication to the author.

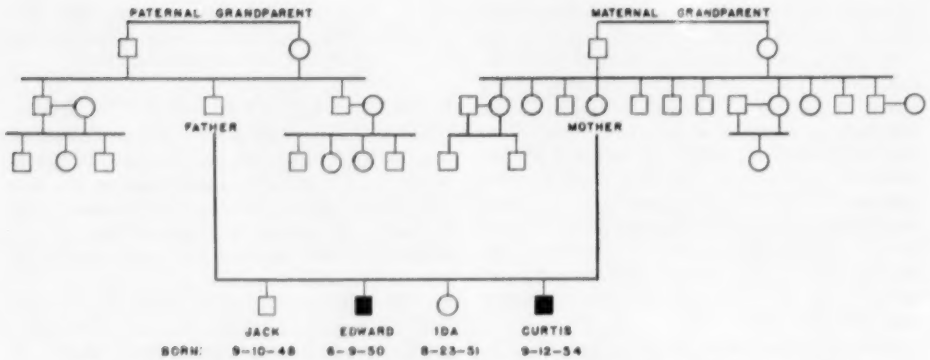

Fig. 3.—The Sandbak family pedigree. The two Sandbak brothers, Edward and Curtis, were operated on for congenital hypertrophic pyloric stenosis in Minot, N. D. Edward's operation was performed on Aug. 22, 1950, at St. Joseph's Hospital by O. S. Uthus, M.D. Curtis was treated surgically on Nov. 10, 1954, at Trinty Hospital.

the Northwest Clinic on Sept. 23, 1951. He was hospitalized the same day at Trinity Hospital, with a diagnosis of pyloric stenosis. Two days later, on Sept. 25, I performed a Fredet-Ramstedt pyloromyotomy using local procaine hydrochloride anesthesia on the typical olive-shaped deformity which was found. Convalescence was uneventful. He was discharged from the hospital Oct. 4, 1951.

Case 3.—Keith, born July 27, 1953, began to vomit when 10 days old. Four days later he was first seen at the Northwest Clinic. This was on Aug. 10. He was admitted to Trinity Hospital the same day with a diagnosis of pyloric stenosis. The next day, Aug. 11, 1953, I performed a Fredet-Ramstedt pyloromyotomy with the patient under local procaine hydrochloride anesthesia on the typical olive-shaped tumor which was found. Recovery followed and the infant was discharged from the hospital five days later on Aug. 16. Figure 2*A* is a photograph of these three brothers taken in January, 1954.

3. The George Sandbak Family (Fig. 3).

The first child, Jack, a normal baby, was born Sept. 10, 1948, when the father was 31 and the mother 21 years old. The second-born child was Edward. His birth occurred June 9, 1950, in St. Joseph's Hospital, Minot, N. D. He did well until the age of 6 weeks, when he began to vomit and to lose weight. Soon afterwards, on July 26, he was admitted to St. Joseph's Hospital under the care of Dr. O. S. Uthus. A diagnosis of congenital hypertrophic pyloric stenosis was made, and medical treatment was instituted. It was not satisfactory, and so surgical therapy was resorted to on Aug. 22, 1950, a Fredet-Ramstedt operation being performed by Dr. Uthus upon the typical pyloric deformity which was found. Death occurred 30 hours later. Autopsy revealed bronchopneumonia and pulmonary edema.

Ida was born Aug. 23, 1951. At the age of 4 months she was first seen at the Northwest Clinic, where she was found to have a cardiac ailment which was diagnosed as Feidler's isolated myocarditis. She died Jan. 14, 1952. Autopsy performed by Dr. Paul Breslich revealed marked hypertrophy of the myocardium and dilatation of the lumens of the ventricles (subacute interstitial myocarditis), chronic interstitial pneumonitis, and subacute endocarditis of the left ventricle.

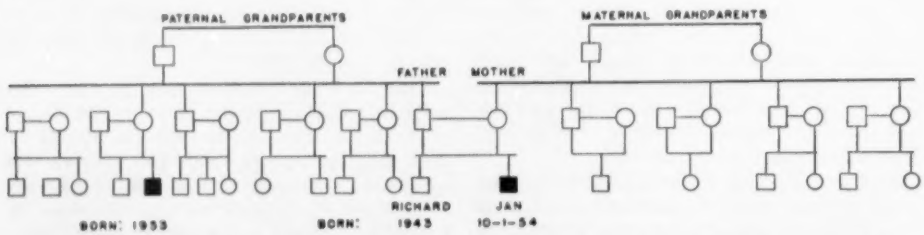

Fig. 4.—The Savchenko family pedigree. Jan Savchenko was treated surgically for congenital hypertrophic pyloric stenosis at Trinty Hospital, Minot, N. D., Nov. 9, 1954. His first cousin, Tulane Peterson, was treated surgically for the same affliction at Abbott Hospital, Minneapolis, in March, 1953, by J. A. Johnson, M.D.

Curtis was born in Trinity Hospital, Minot, Sept. 12, 1954. He was well until a few days before he was admitted to Trinity Hospital, Nov. 4, because of projectile vomiting. The history and physical and x-ray findings were in conformity with the signs and symptoms of congenital pyloric stenosis. He was operated upon for this trouble Nov. 10, 1954, after a short delay because the parents were away on a trip, also because the baby had a mild cold. A Fredet-Ramstedt operation was performed on the typical pyloric deformity which was found. This was done with the patient under local anesthesia, 7 cc. of 1% procaine hydrochloride solution being used. Recovery was prompt.

As the family pedigree (Fig. 3) shows, Edward and Curtis are the only members to be afflicted, not only among four siblings but also so far as the following relatives are concerned about whom there is satisfactory information: parents, grandparents, 12 uncles and aunts, and 10 cousins.

4. The Vance Savchenko Family (Fig. 4).

The son, Jan Savchenko, was first seen in the Pediatric Department of the Northwest Clinic by Dr. R. E. Dormont, Nov. 8, 1954. He was then 38 days old, having been born Oct. 1, in Trinity Hospital, Minot, N. D. Vomiting had begun at age of 4 weeks. Within five days it became forceful. A clinical diagnosis of congenital hypertrophic pyloric stenosis was made, and the child was admitted to Trinity Hospital Nov. 8. The next day a successful Fredet-Ramstedt operation was performed on the typical pyloric deformity which was present. The only other child in this family is an 11-year-old brother, who has always been well. The father and mother are now 37 and 34 years old, respectively. Neither they, their parents, or any of their nine brothers and sisters had any feeding problem in infancy, but one of their 18 nephews and nieces did, a male cousin of their son Jan, Tulane Peterson. He was first seen at the Nicollet Clinic by Dr. W. W. Luek,† in March, 1953, when he was 1 month old. A clinical diagnosis of congenital hypertrophic pyloric stenosis was made and was verified next day at operation, which was performed successfully in Abbott Hospital, Minneapolis, by Dr. J. A. Johnson. The only other sibling in this family, a brother, has always been well.

COMMENTS

A report of this kind, involving five families, is extremely rare. Added to the four families which have come under the care of the Northwest Clinic is that of the Petersons, which includes one of the two affected cousins seen by us. Another very unique feature found among the affected siblings in this report is the number of them in the Stephan group—three brothers with the disease (Table 3). Halbertsma [8] of Holland, had a somewhat similar experience with 32 infants who came under his care during a 14-year period—1922 to 1935, inclusive. Among three families he found two siblings with the disease and "even one with three occurrences."

It is not at all unlikely that the first-born in both the Madsen and the Sheelar group in our series had pyloric stenosis too, but verifi-

† Personal communication to the author.

Table 3.—*Three Siblings in a Family* *

Author	Nationality of Author	Date of Report	Sex	Birth Rank
1. Parker †	American	1927	.....	2d, 3d, 4th
2. McGregor [61]	Canadian	1931	Male, male, ?	4th, 5th, 7th
3. Halbertsma [8]	Dutch	1936	Male, female, male	1st, 2d, 4th pregnancy
4. Donovan ‡	American	1938	All males	1st, 5th, 9th of 12 siblings
5. Ruddock §	American	1944	Male, male, female	.....
6. Cathala and Cler [66]	French	1944	All females	Six siblings in family
7. Adlington [67]	British from Southern Rhodesia	1949	All males	Born consecutively
8. Nafe ‖	American	1954	All males	Born consecutively

* Verification of the diagnosis is known to have been by operation in all three of the infants included in the reports by Parker, McGregor, Donovan, Ruddock, and Nafe. One infant in each of the trios reported by Halbertsma and Adlington was seen at autopsy—no operation. Twins were involved in two of the trios, one in Halbertsma's group and two in that of Cathala and Cler. Donovan's report of three boys in a family did not appear until 1938. In a personal communication he informed me that his report of only two boys in this same family, made in 1992, was an error—that all three had already been operated upon at that time. In another personal communication, only recently received, Nafe informed me that he, too, has now operated upon three brothers, the anomaly being found in one of the six families he reported in 1947, in which two members in each were affected.

† Parker, D. W., in discussion on Ladd.[29]

‡ References 62 and 63; personal communication to the author.

§ References 64 and 65.

‖ Personal communication to the author.

cation is admittedly lacking and so no such claim is made.

When more than one member of a family is affected, they are usually siblings, two in number and born singly, like our Madsen and Sandbak children. In this review, covering a period of 50-odd years, only 59 verified case reports of this kind have been found. They are included in 36 references, both foreign and American.

It is noteworthy that, with few exceptions, the authors of these reports have been clinicians, usually surgeons, with very limited experience with pyloric stenosis of infancy. On the other hand, those who have treated pyloric stenosis in infants by the hundreds have had, as a rule, very little, if anything, to say on the subject of familial occurrence. It is possible that they have failed to detect the anomaly among their clientele. It is true that its disclosure often requires quite an expenditure of time and effort in history taking and pedigree recording which exceeds by far the usual routine.

For many years after Hirschsprung, of Copenhagen, inaugurated the modern era, in 1887, so far as the recognition of congenital-pyloric stenosis is concerned, its familial occurrence was evidently not observed and reported. As we have already learned, Cautley and Dent repeated a little hearsay evidence on the subject in 1902. In the years immediately following, a number of clinicians, mostly German, recorded alleged observations of its multiple occurrence in families. Among them are Freund,[9] Grisson,[10] Heubner,[11] Rosenhaupt,[12] and Ibrahim.[13] They and others too are still quoted extensively in a completely uncritical way, although none of their case reports was verified.

In his textbook, published in 1908, Thomson[14] likewise stated that he had observed several familial occurences, which he probably did, but he failed to prove it satisfactorily with the evident surgical and autopsy evidence which he had in a very high percentage of the 41 cases which had come under his care.

As a contributor in 1912 to the *Practitioner*, London, LaPage[15] said that "there is not conclusive evidence that it tends to run in families, although Dr. John Thomson quotes three instances." And, as we have seen, LaPage was right apparently. But later that same year Ramstedt[16] reported its occurrence in two siblings, upon one of whom he performed his then newly devised pyloromyotomy operation. The other child was not treated surgically. It finally came to autopsy.

As Table 2 shows, this is the first verified case report found in this review of the literature. The second one appeared the next year. It was made by Hammond[17] before the Philadelphia Pediatric Society, in June, 1913. Noteworthy too is the fact that neither of these two early reports came from a surgeon who, at that time, had had any considerable experience with pyloric stenosis of infancy. There was none then, of course, who had many cases to his credit. Hammond had treated only four infants surgically—all with successful posterior gastroenterostomy operations. As has already been noted, only 62 similar occurrences—two siblings in a family—have been noted and verified since, during the intervening 41 years. Fourteen of these observers were Americans, and the rest were mainly British and French. These published observations of two siblings in a family, born singly, have appeared in considerably greater numbers during the last 20 years. They are particularly few during the first 20-year period after 1912 and 1913. Kerley,[18] a New York pediatrician, reported the third undoubted familial occurrence of the disease before the American Pediatric Association, in 1918, and after that there was an interval of eight years before Heekes,[19] of Britain, published the fourth occurrence, in 1926—that is, of two siblings born singly.

It is interesting to note that Richter,[20] of Chicago, who had operated upon 22 infants for pyloric stenosis by 1914, reported that same year that a sister of one of them and a first cousin of another had died of what was clinically the same condition. In making this observation, Richter became one of the few pioneer surgeons with relatively extensive

experience with pyloric stenosis of infancy to note its possible familial occurrence.

Still,[21] of London, had the following to say in 1915 on its "family incidence" in his textbook on "Common Disorders and Diseases of Childhood":

> The question is often asked, when one case has occurred in a family, especially in the firstborn, whether the subsequent children are likely to suffer from the same condition. Formerly I had always replied in the negative but in recent years, amongst a large number of cases, I have seen three instances of two children in a family showing congenital hypertrophy of the pylorus. The rarity of this family incidence is evident, from the fact that only three instances were observed in ninety-one families in which the condition occurred.

Although the cases he cited lacked verification, Still's unusual interest in the subject at this time is quite remarkable. The question he raised then about the likelihood of the disease appearing again among siblings, once one is afflicted, is still unanswered. And his unverified claim of more than two affected siblings, singly born, was not to be the last before an authentic case report of this extremely rare anomaly was reported.

Exclusive of the Stephan family described here, they number only 10 (Tables 3 and 4). In eight instances three siblings were affected, and in two families four had the disease. Four of the observations where three children were affected are by American authors.

Parker,‡ of Manchester, N. H., was the first, apparently, to record a verified anomaly of this kind. In 1926, when discussing a paper by Ladd [22] on pyloric stenosis in 197 infants, Parker reported having observed the disease in 3 of a family of 4 children. His most unusual experience with the familial occurrence of pyloric stenosis aroused no comment whatsoever at this meeting of the New Hampshire State Medical Association except for an exception taken by Ladd concerning the timing of one of the three successful operations on the babies. Finkelstein,[23] in 1921, and Sauer,[24] in1924, made like but unverified claims, the former merely stating that he knew "one family with four and two with three such illnesses" and the latter, equally brief in his report, said that he had "had under his care two families in each of which three cases occurred."

These two extremely brief and unverified statements have received quite universal acceptance, while Parker's report of proved cases is seldom mentioned.

The two families with four children affected in each were described only recently, one in 1948 by Gailey,[25] of Ireland, and the other by Christiaens and Lanthier,[26] of France, in 1951. There were six children in the first family and eight in the second. Among the unverified claims in this connection, which is frequently referred to—and with full approval, too—is that made by Fabricius and Vogt-Møller [27] in 1937. Of the four siblings which these Danish authors reported to be affected, all were treated medically exclusively, with one death. The diagnosis of pyloric stenosis was confirmed in this one child only—at autopsy.

No valid case report involving more than four siblings has been found in this review. It is true that DeLange,[28] of Holland, stated in 1936 that she "once saw a family where five children were affected," but like Finkelstein, Sauer, and others, she failed to prove her assertion—did not try.

After Ramstedt's report in 1912 concerning siblings born singly, approximately 11

TABLE 4.—*Four Siblings in a Family* *

Author	Nationality of Author	Date of Report	Sex	Birth Rank
1. Gailey [25]	Irish	1948	All males	3d, 4th, 5th, 6th
2. Christiaens and Lanthier [26]	French	1951	Male, female, male, male	1st, 2d, 7th, 8th

* These eight infants—seven boys and one girl—were all operated upon successfully.

‡ Parker, D. W., in discussion on Ladd.[22]

years elapsed before a verified occurrence in both members of a set of twins was observed and reported (Table 8). Moore [20] of Dallas, Texas, has the distinction of being the author of this report. It was made before the Southern Medical Association, in Washington, D. C., in November, 1923. A few months earlier, Redlin,[80] in Germany, had described a like occurrence of *Pylorospasmus* in twins, but his diagnoses were unconfirmed either surgically or at autopsy. Moore's paper was published in March, 1924, and in another American journal which appeared that same month Sauer stated that he had cared for twin boys with pyloric stenosis and that his diagnosis was verified after death. Later in the same year, Davis,[81] of Omaha, after reading Moore's article, reported another similar occurrence in twin male infants. They had been operated upon successfully by his father, B. B. Davis, in 1919. They, therefore, appear to be the first twins on whom a diagnosis of pyloric stenosis was made in both and, incidentally, also the only set to live among the first three verified ones to be reported.

After these three sets of twins were reported in 1924, four years elapsed before another contribution of this kind appeared, and since then, during the last 26 years, only 21 more have been found. All told, this review has revealed only 28 sets of twins both of whom were afflicted. This small number is particularly noteworthy here because the twin type of familial occurrence of the disease is the second largest.

Twins have been studied with great interest in recent years by medical geneticists to determine the relative role of heredity and environment in the etiology of the disease when present in either one or both members of a set. Sheldon [82] suggested in 1938 that "a study of the disease in twins should throw further light on the presence of an inherited factor." It is now referred to as the "twin method" of investigation. Its peculiar merit rests on the fact that monozygous twins are, gene for gene, identical and will, therefore, very likely have the same congenital anomalies, such as pyloric stenosis, if it is one; and, conversely, dizygotic twins are not any more susceptible to congenital anomalies than are siblings born singly, since they are no more alike genetically.

The dependability, then, of this kind of an investigation evidently rests, among other things, upon the accuracy of the classification of the sets of twins involved—whether they are monozygous or dizygous. But seldom has this question been answered satisfactorily, with evidence that is at all acceptable. Frequently no statement about it is made, and when an author has committed himself definitely he has usually relied upon the appearance of the birth membranes, but not necessarily as he has observed them. Occasionally a midwife has been asked to recall what she saw, or almost as unreliable information may have been obtained from the family physician. As we now know, an examination of the birth membranes under the most favorable conditions and by skilled examiners still leaves much to be desired. Apparently the question of zygosity, where the twins are of the same sex, must finally rest upon information which is most satisfactorily and reliably obtained after the age of infancy has passed. The material which has been investigated by the "twin method" up to the present time, lacking this kind of zygosity determination, is therefore quite unreliable. However, there is quite universal acceptance of the pronouncement which Sheldon made in 1938 that "the evidence indicates that when pyloric stenosis affects uniovular twins, both infants show the condition" and that when binovular twins are affected "the disease is likely to occur only in one twin." All authors concede, though, as Sheldon did, that there are exceptions to these rules—which, as we have just seen, are based upon impressions only.

Occasionally, relatives other than siblings have been affected. As Carter and Savage [83] have stated recently, "it is not clear that these are more than chance occurrences." So far as parent and child relationship is concerned, some lead should be forthcoming soon from the present crop of children whose parents, like themselves, were saved from death in

infancy by the Fredet-Ramstedt pyloromyotomy operation. Until this miraculous technique enabled the victims of infantile pyloric stenosis to grow up and reproduce in goodly numbers, no revealing evidence of this kind could be hoped for. Until now surprisingly few affected infants, however, are known to have a parent who was likewise stricken in infancy. Tables 5 and 6 reveal that only nine such instances have been found in this review.

TABLE 5.—*Father and Child* *

	Author	Nationality of Author	Date of Report	Sex of Child
1.	Donovan [62]	American	1938	Female
2.	Schaefer and Erbes [3]	American	1948	Male
3.	Carter and Savage [58]	British	1951	?
4.	Carter and Savage [58]	British	1951	?
5.	Fenwick [60]	British	1953	2 males

* In the two instances reported by Carter and Savage there is no verification of the statement that the fathers were afflicted. The father in Fenwick's report was not operated upon in infancy. Hypertrophic pyloric stenosis was discovered in his case at the age of 39 years. He was the father of two afflicted sons. Vinson and Brown reported an unusual case from the Mayo Clinic in 1927, in which a man, treated there for cardiospasm with marked dilatation of the esophagus, later became the father of a son who was operated upon elsewhere for congenital pyloric stenosis when 15 days old.

TABLE 6.—*Mother and Child* *

	Author	Nationality of Author	Date of Report	Sex of Child
1.	Ashton [65]	American	1929	Male
2.	Henderson, Henderson, and Spencer [69]	American	1948	Female
3.	Wood and Smellie [59]	British	1951	?
4.	Keizer [34]	French	1952	Male and female

* In Ashton's case a gastroenterostomy was performed upon the mother in 1906 and a Fredet-Ramstedt operation upon her son in 1929. The report by Henderson and coworkers reveals that the mother had a Ramstedt operation in 1923 and her second child, a daughter, was operated upon for pyloric stenosis in 1948. Likewise, in the case of Wood and Smellie, the operation was on the second child. It is stated that her first child had been treated successfully for the same complaint medically.

Keizer reported that the maternal grandfather of the two siblings was such a feeding problem in infancy that rumination was necessary to subdivide his food. At the age of 62 years, when he was operated upon for cholelithiasis, the surgeon discovered hypertrophy of the musculature of the pylorus and dilatation of the stomach. He diagnosed it as congenital pyloric stenosis. The title of Keizer's report is "Congenital Pyloric Stenosis in Three Generations."

In 1952, Keizer [34] reported from France the only instance I have found in which there is substantial but not entirely satisfactory evidence to support the claim that three generations in a family were afflicted. A mother and her two siblings are known to have had the disease. All three were treated surgically for it in infancy. The maternal grandfather of these siblings had suffered from uncontrollable vomiting in infancy. He was such a feeding problem, so it is stated, that rumination was necessary to subdivide his food. His treatment during infancy was medical. At the age of 62 years a diseased gall bladder was removed, and at this time hypertrophy of the musculature of the pylorus was discovered together with dilatation of the stomach. The stomach condition was diagnosed as congenital pyloric stenosis and Keizer entitled his report "Congenital Pyloric Stenosis in Three Generations."

Nafe reported an uncle-nephew occurrence of the disease in 1947, the uncle being a brother of the child's mother.

TABLE 7.—*First Cousins*

	Author	Nationality of Author	Date of Report	Number of First Cousin Relationships
1.	Cockayne [72]	British	1934	2 *
2.	Thomson and Gaisford [41]	British	1935	1 †
3.	Halbertsma [8]	Dutch	1936	1 ‡
4.	Cockayne [72] §	British	1938	2 ‖
5.	Halbertsmà [73]	Dutch	1949	1 ¶
6.	Carter and Savage [58]	British	1951	1 ‡

* In the first instance the mothers were sisters and the cousins were a boy and a girl. In the second instance the father of one cousin—a boy—was a brother of the mother of the other cousin—also a boy.

† No information about parental relationship. There were two girls in one family and one in the other, and all three had pyloric stenosis.

‡ The father of two afflicted boys had a sister who was the mother of an afflicted brother and sister—four afflicted cousins, then, in this instance.

§ In his second paper (1938) Cockayne [71] reported a third occurrence of pyloric stenosis in two first cousins, but the diagnosis was verified in only one. At the same time, he reported one instance of the disease in two second cousins —both girls.

‖ In the first instance the mothers were sisters and the cousins were two boys. In the second instance the mothers were sisters and the cousins were a boy and girl.

¶ The mothers were sisters and the cousins were two boys. The author, as we have seen, noted four instances of familial occurrences among the first 22 infants he operated upon and only 1 among the next 68, that being this case of cousins.

‡ Among the parents in four families were two brothers and two sisters. In each of these four families one infant had pyloric stenosis—four afflicted cousins then in this instance. The four related parents of these four cousins had seven other brothers and sisters only one of whom was married and was the father of one child and he did not have the disease.

As Table 7 shows, not many first cousin relationships have been found. In reporting pyloric stenosis in four first cousins in 1951, Carter and Savage remarked that "if 200 affected children had 1000 first cousins one

would expect to find a few of these affected even if the true incidence among them was only that of the general population." In the family which they observed, 5 of 11 brothers and sisters had 10 children, 4 of whom were the ones affected. One brother and three sisters were thus involved. The fifth parent in this group of five was a brother, whose two daughters remained well. The one familial occurrence of the disease in first cousins which we have noted among our own group of infants makes only seven such occurrences of bona fide cases of which we are aware except for 11 more instances reported recently by McKeown, MacMahon, and Record,[88] of Birmingham, England. "Of 3980 cousins at Risk, eleven were affected." They added that "the number expected is 11.9, assuming the incidence in cousins to be the same as in the general population of births."

The data which this group published on the familial incidence of pyloric stenosis was based on a "field inquiry for 477 fraternities which contained 489 propositi. (85 per cent of all diagnosed cases domiciled in Birmingham and admitted to Birmingham hospitals in the ten year period 1940-1949)." They accepted diagnoses of pyloric stenosis made upon alleged palpation of a pyloric tumor as well as upon finding it to be present at operation or autopsy. How many diagnoses were made by palpation alone is not stated. They found the incidence in siblings to be 58.1 per 1000 live born infants (male 98.4; female 16.8), which is, as they state, "about nineteen times the incidence in the general population." In all the 14 affected siblings involved in their computations the diagnosis was confirmed at operation.

These authors said too that "the only previous attempt to assess the incidence in sibs of propositi is that by Cockayne and Penrose (1943) who gave the proportion affected as one in twenty, about the same as the estimate recorded above." Of course, a greater tendency of siblings of affected propositi likewise to have the disease is most significant whatever the figure, but if it does actually amount to 19 or 20 times the incidence for the general population it is truly astonishing. Great interest, therefore, will be shown, no doubt, in further investigations of this kind. Confidence in them should be increased appreciably if only verified case reports are utilized for study.

The very small number of verified case reports which we have found can best be explained chiefly on the basis of rarity of familial occurrence or noteworthy failure of clinicians to observe and report the anomaly. Very likely both explanations are applicable. The only improvement in this situation, then, is evidently in the hands of the clinicians. According to statistics previously cited, there

TABLE 8.—*Twins—Both Affected* *

	Author	Nationality of Author	Date of Report	No. of Sets	Sex
1.	Moore [59]	American	1924	1	Male
2.	Sauer [84]	American	1924	1	Male
3.	Davis [61]	American	1924	1	Male
4.	Bilderback [73]	American	1928	1	Male
5.	Cockayne and Sheldon [74]	British	1928	1	Male
6.	Sommer [75]	German	1931	1	Female
7.	Varden [76]	American	1933	1	Male
8.	Redgate [77]	British	1935	1	Male
9.	DeLange [58]	Dutch	1936	1	Male
10.	Ponyanne [78]	French	1938	1	Male
11.	Card [79]	American	1939	1	Male
12.	Robertson [80]	Canadian	1940	2	Male
13.	Ujsaghy [81]	Hungarian	1941	1	Female
14.	O'Donnell and Klein [49]	American	1941	1	Male
15.	Cathala and Cler [66]	French	1944	1	Female
16.	Ladd, Ware, and Pickett [52]	American	1946	1	Not stated
17.	Laubscher and Smith [82]	American	1947	1	Male
18.	Parmelee [54]	American	1948	1	Not stated
19.	Hughes, Small, and Macky [83]	British	1949	1	Male and female
20.	Wagner and Baratz [86]	American	1951	1	Male
21.	Welsh [87]	American	1951	1	Male
22.	Cunningham and Kelly [85]	American	1952	1	Male
23.	Sale [89]	British	1953	1	Male
24.	Metrakos [90]	Canadian	1953	3	2 male; 1 female
25.	Wall [91]	Australian	1953	1	Male

* Although a satisfactory genetic study of familial occurrence of pyloric stenosis in twins requires exact knowledge of the type of twinning in each case—whether monozygotic or dizygotic—no attempt has been made to provide such information here, because in most case reports either no statement is made about it or what is said lacks verification. Satisfactory evidence requires a great deal more information than the obstetricians can give and usually have provided. Of course, a sex difference in a set of twins always means that they are dizygous and no more alike genetically than siblings who are born singly.

TABLE 9.—*Reported Familial Occurrences Wholly or in Part Unverified* *

	Author	Nationality of Author	Date of Report
1.	Armstrong [93]	British	1771
2.	Cautley and Dent [7]	British	1902
3.	Freund [9]	German	1903
4.	Grisson [10]	German	1904
5.	Heubner [11]	German	1906
6.	Rosenhaupt [13]	German	1907
7.	Ibrahim [14]	German	1908
8.	Thomson †	British	1908 and 1921
9.	Still [51]	British	1915
10.	Finkelstein [58]	German	1921
11.	Redlin [60]	German	1923
12.	Feer [64]	German	1924
13.	Richter [66]	American	1924
14.	Sauer [84]	American	1924
15.	Caulfield [93]	American	1926
16.	Monrad [97]	Danish	1927
17.	Bratusch-Marrain [50]	German	1928
18.	Schreiber [98]	French	1933
19.	Thomson and Gaisford [41]	British	1935
20.	DeLange [86]	Dutch	1936
21.	Orgeolet [42]	French	1936
22.	Fabricius and Vogt-Møller [27]	Danish	1937
23.	Brendle [5]	German	1937
24.	Cockayne [71]	British	1938
25.	Lewis [101]	British	1941
26.	Nafe [2]	American	1947
27.	Schaefer and Erbes [3]	American	1948
28.	Todd [99]	British	1949
29.	Grimes, Bell, and Olney [4]	American	1950
30.	Powell and Carter [102]	British	1951
31.	Fenwick [69]	British	1953

* Armstrong's report is of historical interest only. His infant who died of the "watery gripes" at 3 weeks of age was found at autopsy to have a distended stomach, empty intestines, and "a spasm in the pylorus" may well have had pyloric stenosis, but there is no verification whatsoever that the other two siblings did who died at the same age and allegedly "with the same disease."

Cautley and Dent, themselves, never claimed a familial occurrence of the disease in their report of a successful pyloroplasty operation upon an infant with pyloric stenosis. What they did say was that "the grandmother on the father's side lost a sister in infancy whose death was ascribed to nothing passing through the stomach" and they added, in referring to this hearsay evidence, that "we lay no stress on what may be merely a coincidence."

Freund's diagnosis was confirmed at autopsy in only one of two sisters treated medically.

Likewise, Grisson's was verified at operation in only one of two siblings.

Heubner's claims rest solely on hearsay evidence except for "two other cases which I myself could verify" but he didn't say how.

Only one of Rosenhaupt's two siblings had verification, and this was at autopsy.

Ibrahim, in my review of the reference cited here, failed to report a verified case of his own.

Thomson failed to verify his claims made in 1908 and in 1921 by evidence which he evidently had, since a high percentage of his cases came to operation or autopsy.

Still simply stated that he had "seen three instances of two children in a family."

Finkelstein's claim also rests solely on the brief statement which follows: "I myself know one family with four and two with three such illnesses."

The diagnosis in neither one of the twin brothers reported by Redlin was verified. Both were treated medically and recovered.

Feer's evidence rests on his statement that he had under his care an afflicted pair of siblings who had the same great grandfather as did another patient with the disease.

Richter and Sauer both gave brief and unsupported statements, the former reporting familial occurrence of the disease twice in his own experience, and the latter, so he stated, "had under his care two families in each of which three cases occurred."

Caulfield's report of the disease in a father and his two sons was verified in only one of the group, a son.

Monrad simply reported that a family occurrence was found 7 times among 228 cases.

Bratusch-Marrain's report of three affected siblings was verified in only two—one at autopsy and one at operation.

Schreiber's alleged familial occurrence in three generations was supported by valid evidence in only one member.

Thomson and Gaisford did report a verified instance of the disease in two sisters, but they left unverified their statement that in another family two brothers were affected.

Likewise, DeLange, after reporting an authentic familial occurrence of the disease in twins failed to support at all his most unusual statement about seeing "a family where five children were affected with pyloric stenosis."

In four of the five families which Orgeolet reported the case reports were all verified, but in one family verification was lacking in the case of one of two siblings.

The four siblings reported by Fabricius and Vogt-Møller were all treated medically. There was verification of the disease in only one of this group, the one who died and on whom autopsy was done.

In none of the four families which Brendle reported was there verification of familial occurrence. None of his diagnoses and statements was based upon direct observations. The nearest he came to verification of the disease in more than one member of a family was in the first family reported. But here he leaves the reviewer without satisfactory evidence to support his claim that three brothers had the disease. It is evident that two died but that only one was examined at operation.

Cockayne's report is verified for two pedigrees in which cousins were affected, but in a third the diagnosis was not verified in one of the cousins.

Lewis' report of pyloric stenosis in identical twins was unverified in one member of the set.

Schaefer and Erbes have left the reader very much in doubt about the validity of all but one of their nine claims of familial occurrence among 248 pyloric stenosis cases, 232 of whom were operated upon. The one statement they make which leaves no doubt about the validity of the case concerns a father and son who were both operated upon for pyloric stenosis.

Todd reported seeing three siblings with the disease who were treated successfully with atropine methyl nitrate (Eumydrin).

In the 14 alleged familial occurrences of the disease among a group of 119 infants reported by Grimes, Bell, and Olney the authors fail to give any verifying evidence, saying only that "there was a positive family history."

In only one of the dizygous twins reported by Powell and Carter was the diagnosis verified.

Fenwick's report of the disease in a father, his two sons, his nephew, and his brother was verified in infancy in the cases only of the two sons who were operated upon. Hypertrophic pyloric stenosis was found at operation in the father when he was 30 years old. His nephew, in whom a tumor was palpated—presumably pylorie—was treated medically. The suspect brother died in infancy, years before these events, from persistent vomiting.

† References 14 and 94.

must be about 12,000 good occasions yearly in this country alone for an inquiry to be made about a possible familial occurrence.

SUMMARY AND CONCLUSIONS

Four instances of familial occurrence of congenital hypertrophic pyloric stenosis are reported. They were observed at the Northwest Clinic, Minot, N. D., during the last 17 years among an unselected group of 68 infants, all of whom were treated surgically for the disease. Siblings were affected three times and first cousins once. In one family of four brothers the three youngest had the disease.

The world literature is reviewed quite extensively. Fewer than 125 verified familial

occurrences have been found. They are listed here in Tables 2 to 8, inclusive. A listing has also been made in Table 9 of the reported familial occurrences which remain wholly or in part unverified.

The incidence of this anomaly is unknown, but it appears to be extremely rare. Probably all reported cases, whether authentic or not, would number not more than 150 to 175.

Siblings alone are involved in the relationship in approximately 75% of cases. Usually two male infants, singly born, constitute the affected family group. Twins are next in frequency, but their number is small, only 28 sets having been found in which both members were afflicted. Because of doubtful zygosity in most instances, their utilization in the study of the possible genetic factors involved in the etiology of the disease is not at all encouraging for trustworthy findings and conclusions.

As yet it remains to be proved that parents who have had pyloric stenosis in infancy are more likely on this account to have children with the disease. In the very few recorded instances in which they have had affected offspring the factor of coincidence must be considered. If a genetic factor does play any part in the development of the disease, there is hardly any evidence that it reveals itself in three generations. There is no evidence either that parental consanguinity has been an etiological factor.

The place, if any, which the genetic factor has in the etiology of congenital hypertrophic pyloric stenosis has not been discovered yet and, for reasons already stated, probably will not be for many years to come—not until large numbers of verified familial occurrences of the disease have been reported in sufficient detail for the medical geneticists to utilize.

DISCUSSION

Dr. George B. Packard, Denver: Dr. Cameron has presented a very interesting subject, of which he has made a most careful study. After reading his outline in the program, I decided to review our cases for comparison with his experience.

In a recent review of 241 personal cases, of which 118 were not the first-born, we have the following figures. Four had brothers who had had the same operation for pyloric stenosis. One had a brother who was said to have had the same symptoms but who had died without an operation. In two cases, it was reported that an older brother had had very severe similar symptoms which had cleared up under medical care.

We had no case of three siblings, as Dr. Cameron has reported. There was one child whose mother stated that she had been operated on for the same condition about 24 years previously at the Children's Memorial Hospital, in Chicago. Incidentally, this was her second child, not her first.

As something was said about treatment of pyloric stenosis, I might just mention that a recent review of the last 300 cases of this disease at the Denver Children's Hospital has been made by Dr. David Akers. He found a trend in treatment which differs from the procedure of several years ago. While in the textbooks and in most former articles it has been stated that these babies should be observed for some time, that they must be given a fair trial with medical treatment before surgery, and that the tumor must be found, the tendency here now is to handle these patients much more quickly. In most cases, the final diagnosis has been made before the babies are brought to the hospital.

Practically all of our cases at the Denver Children's Hospital are x-rayed, and the x-ray shows so positively whether or not there is a pyloric stenosis of the congenital type that the decision to operate is made almost from the x-ray, regardless of palpation of tumor.

In fact, when the pediatrician has a patient who is not doing well and whom he suspects of having a pyloric stenosis, he will send the baby for x-ray and then will call the surgeon and say, "I have a patient with pyloric stenosis." That patient then goes to the hospital.

It takes 24 hours to determine and correct his electrolyte balance, so most of these babies that have a true pyloric stenosis are diagnosed early and are operated upon about 24 hours after admission.

Dr. Everett P. Coleman, Canton, Ill.: I want to give briefly one other case report.

Some years ago a boy was born. He had apparently some malnutrition. He did a lot of vomiting and finally died. Two years later his mother had a daughter. The same thing happened to her. This time the mother was more alert, and the patient was seen by Dr. Clifford Collins of Peoria, formerly a member of this Society.

Dr. Collins did the Ramstedt operation, and the girl survived. She grew up and married and had two boys. I have operated on both of these children. Each one developed the same sypmtoms of pyloric stenosis, but this time an alert grandmother made the diagnosis, and they were brought to surgery quite early. Both survived and are still alive.

I just wanted to report this as an additional family for Dr. Cameron's information.

DR. J. WILLIAM THOMPSON, St. Louis: I thought Dr. Cameron might be interested in this, as well as the rest of the Association: My brother, Gershom, who is also a member of this Association, undoubtedly had the symptoms of pyloric stenosis when he was an infant. His first two children had this disease and were operated upon by members of this organization, one by Dr. Waltman Walters and the other by Dr. Charles W. Mayo.

DR. EUGENE A. OSIUS, Detroit: I was very much interested in Dr. Cameron's paper. I have a few figures that I should like to quote. You haven't heard many figures today, so I will add a few more.

It is interesting to note what these figures comprise. They come from the surgical services of the Children's Hospital of Michigan and Harper Hospital, Detroit, and are a collective review of patients who have been operated upon by the following men: Dr. C. D. Benson and Dr. Grover Penberthy, both members of this Association, who have done the major number of these cases, also, Dr. C. E. Weller, Dr. E. Lauppe, Dr. C. E. Maguire, and myself.

Included in this are the cases that we have allowed the residents to do under our supervision, a rule that we are extremely strict in enforcing. We feel it works for the benefit of the resident and the hospital as well as the patient.

Between 1940 and 1950, 517 patients with congenital pyloric stenosis were operated upon; 425 were males and 92 were females. There was a mortality of four, which is 0.7%. One was a wound disruption, one was a wound disruption plus intestinal obstruction, one was a bronchopneumonia, and one was that bugaboo of all pediatric wards, diarrhea.

We have a population in Detroit of around 2,000,000; about 400,000 are Negro, but we see relatively few Negro infants with pyloric stenosis. There seems to be a slight increase in the last few years, but it is not very marked.

The age at the time of the onset of symptoms is interesting, that is, the recognition by the pediatrician or whoever sees the patient first. From 1940 to 1944 it was 22 days; from 1945 to 1950 it was 16 days, a difference of 6 days, which I think is quite significant and speaks well for the pediatricans and for the men who see the cases first.

The preparation preoperatively has been reduced on an average from four to two days, and the stay in the hospital has been reduced from eight to four days on the average.

As far as the genetic set-up is concerned, there were four sets of twins. The first two sets had four operations, one each (no partiality shown). In the next two sets there were only two operations. We lost out a little bit there. They were all boys.

Just before I left Detroit, about there weeks ago, I operated on a boy for pyloric stenosis with a large tumor and with a good postoperative visit result.

The father of my patient had had two first cousins who were operated on for pyloric stenosis, congenital.

I have enjoyed Dr. Cameron's paper very much. The viewpoint is interesting. In the future we will pay more attention to this feature.

DR. ANGUS L. CAMERON, Minot, N. D.: I want to thank the discussers of my paper. Dr. Thompson's report concerning his brother and the latter's two children, all three of whom had congenital pyloric stenosis, is unique so far as I have been able to determine from quite an extensive review of the literature. I have found nine reports of a parent and one child having had the disease but none in which two siblings and a father or mother were afflicted. I do not doubt that there are a number of unreported cases of familial occurrence involving a parent and child, but the evidence indicates that the disease rarely afflicts two generations. We therefore cannot look upon it as having a strong hereditary tendency between parents and their offspring.

I found in my review of cases recorded in the literature that the subjecting of a baby to operation for pyloric stenosis does not always mean that it had the disease. To my surprise I learned that some present-day surgeons advocate and practice some kind of a Fredet-Ramstedt operation on infants erroneously diagnosed preoperatively as having pyloric stenosis. The finding of the characteristic pyloric deformity at operation or autopsy is the only absolutely reliable evidence that the disease exists.

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Unusual Tumors and Cysts of the Neck

ARNOLD S. JACKSON, M.D., Madison, Wis.

In operating upon tumors and cysts of the neck one may expect to encounter at least 50 different pathologic conditions. These may comprise the common lesion, such as adenomatous goiters, mixed tumors of the salivary glands, and benign conditions, such as lipomas, fibromas, and sebaceous cysts; branchial and thyroglossal cysts, and the metastatic and inflammatory lesions.

In the course of many surgical neck procedures, sooner or later some unusual pathologic entity is sure to be encountered, and often enough it is the pathologist and not the surgeon who first becomes aware of the rare find.

In the brief time of 10 minutes allotted for these discussions it is only possible to mention a few of the seldom-encountered tumors and cysts which I have operated upon during the nearly 30 years that I have been a member of the Western Surgical Association.

MIKULICZ'S DISEASE

At the Fifty-Eighth Meeting of the Association I presented a report of 2 cases of Mikulicz's disease, of which there are less than 100 authentic cases in the American literature. At the time of this presentation only one of our members stated that he had observed such a case. Since this time I have had a third case and have thus fulfilled the saying that rare lesions usually come in threes. It is interesting to note that the three cases were successfully treated by three different methods, the first by penicillin, the second by surgery, and the third by x-ray. One should note that most of the cases of so-called Mikulicz's disease reported in the literature do not fulfill the diagnostic criteria as laid down by Mikulicz in his classic description of the disease given by him before the Society for Scientific Medicine at Königsberg in 1888. Briefly, he decreed the following triad of signs and symptoms as diagnostic of the disease: (1) symmetrical enlargement of the salivary glands, (2) narrowing of the palpebral fissures, and (3) parchment-like dryness of the tongue. Microscopic examination shows a replacement of glandular tissue by lymphoid tissue.

Fig. 1.—Calcified branchial cyst (preoperative).

From Jackson Clinic and the Frieda Meyer Nishan Foundation.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 4, 1954.

Fig. 2.—Gross specimen.

It would be interesting to ascertain if any members of the Association have encountered any case of true Mikulicz's disease since the report presented to the Association in 1950. This condition should not be confused with the multitude of reports listed in the literature under Mikulicz's syndrome, which includes many diseases such as tuberculosis, syphilis, and leukemia.

SARCOMA OF THE THYROID

During the past three decades great interest has been manifested in diseases of the thyroid gland and, in particular, in malignant disease. This interest has been stimulated by the increasing number of reports and the apparently increasing incidence. Our members, notably Cole, Hunt, and Black among others, have been among the foremost to stimulate interest in this subject, and two papers are listed on this subject on today's program. Cole created something of a sensation when he reported some years ago an incidence of 12% malignant growths in the multiple and twice in the so-called clinical solitary adenoma type. Yet I was recently informed that Catell has now observed a considerably higher incidence. It is true that these alarming figures do not truly portray the normal incidence for the surgeon at large, since cases of malignant thyroid disease naturally gravitate to thyroid clinics. In 1949 in a study of 40 cases of malignant conditions of the thyroid, I reported 3 cases of sarcoma of the thyroid, an extremely rare type of lesion. In fact, this condition was so seldom seen that for many years various authors stated that this pathologic entity did not exist. Both of my cases were inoperable, both occurred as malignant degeneration in men past 50, and both diagnoses were confirmed by biopsy. Both responded poorly to x-ray therapy, and neither survived a year. Yet both might possibly be alive today had prophylactic thyroidectomy been performed anywhere from 1 to 20 years before admission. Under the goiter-prevention plan which I established for the state of Wisconsin 20 years ago, I believe most adenomatous goiters and, in turn, malignant degeneration, such as the sarcomatous lesion, can likewise be prevented.

Fig. 3.—Postoperative result.

The rarity of this lesion may be judged from a study on the subject of malignant lesions of the thyroid presented before the American Association for the Study of Goiter by Pemberton in 1938, in which he reported 4 instances of sarcoma in a total of 774 cases.

HÜRTHLE-CELL CARCINOMA OF THE THYROID

Since two papers are to be presented this morning on the subject of carcinoma of the thyroid, I will discuss only the extremely rare Hürthle-cell carcinoma. In a series of 51 cases of malignant lesions of the thyroid, I have operated upon only 2 of these cases. There is nothing characteristic about the gross appearance of these tumors, which appear to be encapsulated, benign adenomas and are consequently seldom removed completely at the time of the primary operation. Considerable difference of opinion exists concerning the degree of malignancy of these tumors, and this degree depends upon the type of cell. The chance of cure is good if the tumor is removed. X-ray therapy does not appear to be effective. Radical neck dissection is indicated only for recurrent or residual tumors. This tumor can metastasize widely, although it resembles a benign adenoma clinically. Radioactive iodine does not appear to be effective. My two cases occurred in middle-aged men; one patient survived five years, and the other, four years, dying from a concurrent lymphoma.

Fig. 4.—Hemangioendothelioma of submaxillary gland (preoperative).

Fig. 5.—Hemangioendothelioma of submaxillary gland (preoperative).

A still more unusual case was that of a malignant endothelioma. In 1926, I operated upon a young woman, performing a subtotal thyroidectomy for a supposedly benign adenoma. When the pathological diagnosis was established, she was given radium therapy. She remained well until 1948, when a recurrence developed, and deep x-ray therapy was instituted. Her subsequent course has not been ascertained.

CALCIFIED BRANCHIAL CYST

Branchial cysts are a not uncommon lesion of the neck, but the following is the only case of its kind I have observed.

In 1950, a man 21 years of age was referred because of a tumor mass on the right side of the neck that was bulging from beneath the sternocleidomastoid muscle. From its location it should have been a branchial cyst, but, because of its extreme hardness and immobility, this condition was not considered. Instead it appeared to be definitely a malignant lesion firmly attached to the jugular vein

Fig. 6.—Operation showing mass extending into mediastium.

and carotid arteries. The relatives were advised that its successful removal and cure did not seem probable but that an attempt would be made. After a difficult 90-minute operation a large hard mass measuring 3 in. by 2 in. by 2 in. was freed from the deep vessels and successfully removed. Much to my gratification, the mass proved to be a calcified branchial cyst; the patient was cured and is now about to be ordained as a priest.

This case illustrates the point of how easy it is to be mistaken in our attempted diagnoses of lesions of the neck, how, despite a rather hopeless appearing condition, the error may be in favor of the patient.

LIPOMA OF THE NECK

Of all the conditions in the neck to be encountered, this is one of the commonest and easiest to dispose of, and yet the following illustrates another case in which I erred in the diagnosis.

A woman, aged 54, came to the clinic complaining of a soft cystic mass in the front of the neck that had been there as long as she could remember, yet only recently had begun to grow. At times it seemed to swell up and become tender. After a much too cursory examination (I was probably hurrying to get away to this or some other meeting), I assured her that, although it was rather a tricky operation to remove a thyroglossal cyst duct, it could be done without serious risk and would require 30 minutes or more. Much to the patient's surprise and my own, surgery took but 10 minutes for the removal of a lipoma that was almost, but not quite midline in location.

Had I observed more carefully, this deflection might have been detected and the correct preoperative diagnosis suspected. The older a surgeon gets, the more humble he becomes.

HYGROMA

This congenital multilocular cyst is benign, of lymphatic origin, and not extremely rare. Still, many good general surgeons have never operated upon one. One of our members, Dr. Kenneth Sawyer, gave an excellent paper on hygromatous cysts before the 1950 meeting. Such cases tend to gravitate to the large children's hospitals and undoubtedly in the future will be cared for by a new variety of specialists, namely, pediatric surgeons, as proposed at our last meeting by Dr. Willis Potts. I have operated upon only three patients, and, in my opinion, the operation is not a simple procedure. To remove these cysts successfully one must dissect finger-like projections of tissue-paper-thin walls extending down into the mediastinum of infants or very young children, and the operation requires time and careful dissection. In some cases rapid growth may cause respiratory difficulty and require immediate surgical relief.

BOECK'S SARCOID

Boeck's sarcoid is neither common nor easy to diagnose clinically. In fact, the diagnosis must be established by biopsy. It may affect the salivary glands as well as other

Fig. 7.—Postoperative result.

organs of the body. Recently I removed a huge spleen in a patient suffering from this disease because of severe anemia and pressure on other organs. A review of five texts on pathology revealed no report of this type. Occurring as it usually does in the neck, sarcoidosis may be mistaken for Hodgkin's disease; however, it runs a fairly benign course, and patients live for many years. Neither its etiology nor its treatment is understood. My experience has been limited to three cases, two in young adults and one in a middle-aged woman. These patients are all living and well, one having survived 20 years since operation.

DERMOID CYST

The dermoid cyst is occasionally found in the neck and, like these cysts elsewhere in the body, it often contains hair and is soft in consistency. It is more superficial than the branchial cyst and is not attached to the lateral pharyngeal wall. An unusual case that I am reporting here is one that occurred in the floor of the mouth, causing such elevation of the tongue that the patient, a young woman, could swallow only with difficulty.

Fig. 8.—Cystic hygroma (preoperative).

Fig. 9.—Gross specimen.

Dissection and removal of this cyst were not difficult and afforded the patient complete relief. This cyst caused a large swelling in the neck, just underneath the chin, giving somewhat the appearance of a thyroglossal cyst.

BRANCHIOGENIC CARCINOMA

Branchiogenic carcinoma is a relatively uncommon sequela of a branchial cyst, and, in a series of over 50 cases of the latter, a malignant lesion was noted in but 2 instances. In thyroglossal cysts malignant change is rare, and I believe the first case was reported in the *New England Journal of Medicine* a year ago. Both of my cases occurred in elderly persons of each sex, and survival was less than two years in both instances. In view of this rare but possible serious complication, patients should be advised to have a branchial cyst removed when diagnosed.

BRANCHIOGENIC FISTULA

Branchiogenic fistula, while not a cyst or tumor of the neck, deserves mention as a

Fig. 10.—Postoperative result.

rare surgical condition. A youth of 16 came to me complaining that at times he had a bitter taste in his throat and that also he occasionally noticed a slight discharge from a small opening in the skin above the clavicle. At the time I had never observed this lesion, but, on consulting the excellent text by Ward and Hendricks on tumors and cysts of the neck, I found a report of this condition. They advise against injection of these tracts with any type of dye and advise using the Hamilton Bailey so-called stepladder method of surgical excision, consisting of multiple transverse incisions somewhat similar to the plan of stripping varicose veins. However, in my case I preferred to cone out the fistulous tract through a longitudinal incision, which I believe is easier to perform. The resulting scar was quite satisfactory.

METASTATIC TUMORS

Metastatic tumors of the neck are not infrequent, and one must always suspect this possibility, especially in persons of the cancer age. The primary tumor may be within the urogenital, gastrointestinal, or respiratory tracts. It may be found in the pharynx, oral cavity, larynx, lips, sinuses, or skin. In my experience there have been two cases in which a metastatic lesion appeared to develop in nodular goiter from carcinoma of the breast, and, if for no other reason, I believe that nodular goiters, when present, should be removed within three months after a radical mastectomy.

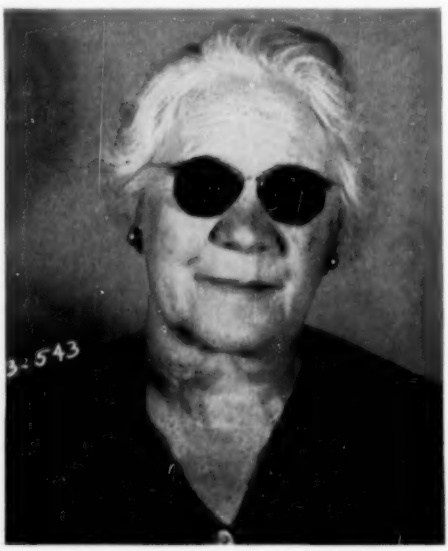
HEMANGIOENDOTHELIOMA OF THE SUBMAXILLARY GLAND

Of the unusual neck tumors I have removed, hemangioendothelioma of the submaxillary gland is undoubtedly the rarest.

Report of a Case

T. L., a boy, was first seen on May 31, 1947, at the age of 6 weeks with no abnormalities noted. On June 20, 1947, the parents noted swelling of the left cheek. Three days later this was seen by a pediatrician, who believed that the infant had a parotitis. A course of penicillin was given, and the mass became somewhat smaller. It soon became large again, and a course of sulfonamides was given, with practically no effect. On July 14, 1947, the patient was seen in surgical consultation, at which time the mass seemed to arise posterior and inferior to the left parotid gland, and the possible diagnosis of cystic hygroma was considered. The mass was followed for six weeks and grew steadily until it

Fig. 11.—Malignant endothelioma. Operated upon in 1926. Recurrence in 1948. Treated with deep x-ray therapy.



was about 8 cm. in diameter. It felt cystic and could not be transilluminated. The mass seemed to lie in the subcutaneous tissues of the neck.

On Aug. 19, 1947, the tumor was removed with the patient under ether-oxygen endotracheal anesthesia. There was a large wine-colored tumor, about 6 by 5 by 4 cm., involving the left side of the neck from the transverse processes of the sixth cervical vertebra, the carotid and jugular vessels, and the anterior surface of the sternocleidomastoid muscle to the skin. The tumor lay adjacent to the submaxillary and parotid glands but grossly seemed to be distinct from them. Many blood vessels extended from the tumor into surrounding tissues. Two lymph nodes were found in the area of the tumor. The tumor was excised intact.

The postoperative course was uneventful, except for development of a partial, transient facial paralysis.

The following pathologic report by Dr. Etheldred L. Schafer gives the gross and microscopic appearance of the specimen removed at surgery: "The gross specimen was a soft but not fluctuant, well encapsulated, dark red ovoid mass, measuring 5.5 by 4.5 by 3.5 cm. The external surface was faintly bosselated. The capsule was thin and transparent. Cut surfaces were coarsely lobulated, dark red, and slightly bulging. A small amount of blood exuded from the surface. Attached at one pole was a separate ovoid mass, measuring 1.5 cm. in maximum diameter, which had pinkish-gray cut surfaces and resembled a lymph node.

"Numerous sections from the mass showed a vascular tumor involving salivary gland tissue. Some of the gland was visible at the margins of the tumor, and isolated glands and ducts occurred throughout the tumor. Delicate fibrous connective tissue septa divided the tumor into lobules reminiscent of the normal pattern of the salivary gland. In some regions, variable-sized, blood-containing spaces with only thin walls separating them were numerous. These spaces were lined by endothelial cells, which in some instances were plump. An occasional mitotic figure was noted among these cells. In other foci, compact masses of small cells occurred between the blood spaces. These cells had indistinct cell outlines. Their nuclei were round or irregularly oval and fairly uniform in size. The chromatin material was irregularly dispersed throughout the nucleus; none contained a nucleolus. Mitotic figures were more numerous in these compact foci, numbering as many as 4 to 5 per high power field. An occasional multinucleated cell was noted. The lymph node at the periphery of the tumor was normal.

"The tumor belongs in that group of tumors known as angiomata, specifically to the hemangiomata. The following must be considered in making a diagnosis: hemangioma (capillary type), hemangioendothelioma, hemangiopericytoma, and hemangioendotheliobla stoma. The exact nature of the tumor cells and their relationship to blood vessels could not be demonstrated without special stains. Sections stained for reticulin by the Laidlaw method revealed anastomosing blood vessel channels with neoplastic cells largely confined within their reticulin sheaths. Thus, they are neoplastic endothelial cells, and the tumor is considered a hemangioendothelioma. Stout, who saw the sections and made the Laidlaw stains, concurred in this diagnosis. Several other pathologists who reviewed the sections variously diagnosed the tumor as a simple hemangioma or a hemangioendothelioma. The extreme cellularity of the tumor, the mitotic figures, and the multinucleated forms within the neoplastic cells remove this tumor from the group of simple capillary hemangioma. The presence of mitoses is thought to be consistent with a period of rapid growth of the tumor in an infant and not to indicate a malignant neoplasm.

"The diagnosis is a hemangioendothelioma arising in the salivary gland."

In a review of American literature from 1916, no mention of this type of tumor arising in the salivary glands was found. Such a case had not been reported at the Children's Hospital, Boston, or was it diagnosed prior to this case in any of the 40,000 children born at the Boston Lying-In Hospital. Other tumors in this region are also rare. The sections from this case have been reviewed by a number of pathologists, all of whom concurred in the opinion that this was a benign vascular tumor; however, it was variously designated as a hemangioma (simple type) and a hemangioendothelioma. Three of the above pathologists mentioned had seen similar tumors, apparently primary, in a salivary gland. No reports have appeared in the literature.

Reading on this subject is difficult, because the criteria for the classification of blood vessel tumors have not been clearly defined. Each author who has written on the subject has his own classification and terminology. Ewing, Geschickter, Watson and McCarthy, and Stout are among those who have classified these tumors.

From a surgeon's standpoint the pathologic interpretation of the tumor is important primarily from a prognostic aspect. Diag-

nosis in this case rests upon specific criteria set up by Stout, which are as follows:

> No tumor should be considered an hemangioendothelioma unless the following two features are present: first, the formation of atypical, endothelial cells in greater numbers than are required to line the vessels with a simple endothelial membrane; and, secondly, the formation of vascular tubes with a delicate framework of reticulin fibers and a marked tendency for their lumens to anastomose.

Stout considers this a malignant tumor except in infants, when it is usually benign.

Hemangioendotheliomas in the necks of infants have been reported by Smith, Watson and McCarthy, and Sweitzer and Winer. None of the three cases reported arose in salivary tissue. The tumors in the case reports of Watson and McCarthy and Sweitzer and Winer were cutaneous and red.

CLASSIFICATION

In summarizing the subject of unusual tumors and cysts of the neck I should like to present a classification that I developed and presented at the Eighth International Congress of the International College of Surgeons, at Madrid, in 1952. No classification ever has proved, or probably ever will prove, satisfactory to those interested in this subject. The one which I presented is compiled from the work of others as well as my own and is offered to aid those interested in the subject.

CLASSIFICATION OF TUMORS AND CYSTS OF THE NECK

1. Tumors of the thyroid and parathyroid glands
   - Thyroid: adenoma, carcinoma, sarcoma
   - Parathyroid: adenoma, carcinoma
2. Tumors of the salivary glands
   - Mixed tumor
   - Primary malignant neoplasm
   - Cysts
3. Primary tumors of the lymph glands
   - Hodgkin's disease
   - Lymphosarcoma
   - Lymphoepithelioma
   - Leukemia
   - Endothelioma
4. Secondary tumors of the neck
   - Metastasis from the buccal cavity
   - Direct extension from the buccal cavity
   - Metastasis from below the clavicle
5. Benign tumors
   - Lipoma
   - Myoma
   - Dermoid
   - Chondroma
   - Hemangioma
   - Lymphangioma
   - Atheromatous cyst
   - Sebaceous cyst
   - Fibroma
   - Adenoma
   - Keloid
   - Cylindroma
   - Xanthoma
6. Unusual tumors
   - Mikulicz' disease
   - Carotid body tumor
   - Neuroblastoma
   - Neurofibroma
   - Ganglioneuroma
   - Boeck's sarcoid
7. Cysts and fistulas
   - Thyroglossal
   - Branchial: benign, malignant
   - Hygroma
8. Inflammatory lesions
   - Nonsuppurative lymphadenitis
   - Suppurative lymphadenitis
   - Tubercular lymphadenitis
   - Diffuse cellulitis (Ludwig's angina)
   - Actinomycosis
   - Blastomycosis
   - Sporotrichosis

SUMMARY

A brief report is presented of 11 unusual surgical lesions of the neck.

Included are various types of carcinoma, sarcoma, huge adenomata of the thyroid, carcinogenic branchial cyst, Boeck's sarcoid, Mikulicz's disease, hygroma, metastatic tumor, hemangioendothelioma of the submaxillary gland, dermoid cyst of the floor of the mouth, and a calcified branchial cyst.

An extremely rare case of hemangioendothelioma is reported in detail.

My classification of tumors and cysts of the neck is presented.

DISCUSSION

DR. PAUL A. KNEPPER, St. Joseph, Mo.: I wish to add one unusual neck tumor to Dr. Jackson's excellent collection. This is a desmoid tumor of the neck. Desmoid tumors are fibroblastic and benign, and probably all of you have seen them in the abdominal wall of women who have had pregnancies.

A somewhat similar condition is that of Dupuytren's contracture or a contracture of the planter fascia. Desmoid tumors of the neck are quite rare. They also are known as congenital wryneck or congenital torticollis, or several other particular terms.

In the past they have been attributed to difficult breech deliveries. However, that theory does not have much support at the present time, and in the slides that I will show you you will notice that there is no pigment or hemorrhage in the area.

The case is that of a 10-day-old infant who was referred to me. The child had had a normal, cephalic presentation. Apparently the tumor was not noticed at birth, but after about five days the tumor was noticed and the mother was sure that the tumor was rapidly increasing in size.

[Slide] The mass was in the right sternocleidomastoid muscle and about 3 cm. in diameter. The area was excised along with the muscle. You see on the left side the normal muscle and a rather encapsulated tumor with some degenerated muscle, but with well-differentiated cells.

A frozen section showed the lesion to be benign. A rather large area was excised. Surprisingly enough, these desmoid tumors do not recur even if they are incompletely removed. This power is $\times$ 50.

[Slide] This is a high power, of $\times$ 200, and, again, it shows some of the degenerated muscle and the well differentiated cells. Again I would like to point out that there is no hemorrhage nor pigment.

DR. JAMES W. HENDRICK, San Antonio, Texas: Dr. Arnold Jackson is to be congratulated on this very timely, interesting, and informative study. We all know of the tremendous amount of excellent work that he has done throughout the years in surgical problems of the neck.

Even with a very wide experience over a period of many years, I am frequently confused as to the exact diagnosis when the patient is first examined in the clinic with a tumor in the neck, and it is not uncommon that I make an erroneous clinical diagnosis.

It has been our teaching that a good clinical history on patients with neck tumors is most important. In general, if the patient has a history of a tumor for a period of years in most instances it is of congenital origin. If it is of several months' duration, most frequently it is neoplastic, and if it is of very short duration, that is, three or four weeks, more probably it is inflammatory. From a review of some 1200 consecutive tumors and cysts of the neck, I confirm the statement of Dr. Jackson that of neck tumors developing after 50 years of age 50% represent metastasis either from above or below the clavicle.

It seems superfluous to stress repeatedly that adequate biopsy of all tumors and cysts of the neck is imperative, remembering that the pathologist can pass on only tissue submitted to him. Consequently, it is essential that representative material be obtained. We see so many patients with tumors of the neck referred for radiation therapy by their family physician without histological study of the tumor. This practice is to be strongly condemned.

To illustrate the difficulty in making an accurate diagnosis and to show three consecutive mistakes that I made from a clinical standpoint and to show also how unusual neck tumors may present themselves, I would like to present cases illustrated with slides.

[Slide] This patient was a 45-year-old woman who came in with a history of a tumor of several years' duration on the left side of the neck that was movable. It was located midway, as you observe, between the angle of the mandible and the clavicle, beneath the anterior border of the sternomastoid muscle. It was movable laterally, firm but not hard. There was a slight thrill associated with it. The patient was in excellent health otherwise. There were several provisional diagnoses. To our surprise when it was exposed at operation, it was a carotid body tumor. When I exposed it, I found that it was located, as all carotid body tumors are, in the bifurcation of the common carotid artery. That was the first time in my experience that I had observed that the common carotid artery may bifurcate any place from the clavicle to the base of the skull; also to my amazement, this anomaly was reported in several anatomies on the subject.

In removing carotid body tumors, we frequently place a bulldog clamp on the common carotid artery, another on the internal, and a third on the external carotid, which we can leave on for a period of 20 to 30 minutes, without deleterious effects. This permits easy dissection of the tumor from the carotid vessels.

The second patient is a woman who was referred with a tumor located in the posterior triangle of the neck with a history that it would enlarge and decrease in size at intervals. The patient stated that frequently it would enlarge following the intake of fluids and food and complained of a foul breath. She was first observed after a morning meal, and the tumor was the size of a lemon and rather firm, and on examining the patient I noticed it decreased in size, after which the patient expectorated a large amount of foul material. A barium swallow demonstrated that the patient had a large diverticulum of the cervical esophagus. At operation the diverticulum was found to have dissected a path into the posterior triangle of the neck and was presenting itself posterior to the sternomastoid muscle. This was my first experience with cervical esophageal

diverticulum presenting a tumor in the posterior triangle of the neck.

The third case is a young man that came in with a tumor on the left side of the neck, just above the clavicle. The tumor had been present eight or ten weeks, measured 4 or 5 cm. in diameter, and was firm and movable. There had been no weight loss. The patient was otherwise in good health. He had a history of having a lymph node removed from his right groin about eight years previously which had been diagnosed as lymphosarcoma. We were unable to obtain the pathologic slides for study. The inguinal nodes had been treated with radiation therapy, the quantity not known. The tumor was removed, and the pathologist reported it as metastatic carcinoma probably from the testis. Careful examination of the testes revealed a primary carcinoma of the left testis less than 1 cm. in diameter.

Dr. Claude J. Hunt, Kansas City: Dr. Jackson has a well-founded reputation as a thyroid investigator and a thyroid surgeon. He has now extended this to a position of authority on tumors of the neck.

I would like to discuss this from the point of view of congenital malformations of lesions that develop in the neck. They naturally have a definite relationship to the embryological development of the neck. I don't think it is amiss to make a few remarks concerning the embryology of the neck.

[Slide] Between the fourth and seventh week in fetal development the neck structures are laid down, and if one studies carefully the embryology of the neck he wonders why there are not more malformations than actually present themselves, because it is a very complex procedure that takes place in the formation of the neck.

As you know, there are clefts and arches that grow forward laterally and fuse in the midline. Each segment is destined to form certain neck structures. These develop along with pharyngeal pouches which contribute to essential structures of the neck.

The first arch, for example, the mandibular hyoid arch, is destined to form the external ear and lower mandible. At the same time that these arches and clefts are growing forward, there are pouches in the pharyngeal structures of the neck that are growing laterally and anteriorly and will eventually fuse in the midline, similar to the clefts and arches. The first pouch forms the Eustachian tube and middle ear. This occurs after the oral cavity, which was originally a solid structure, has become a tube-like structure.

Thus, one layer of epithelial structure which is to form the pharynx grows along simultaneously with the external arches and clefts. If there is any malformation or failure of fusion taking place, there can be a development of a cyst, which occurs in an area of weakness between the pouches and the arches which ultimately will show up in the neck, usually anterior to the sternomastoid muscle, as a branchial cyst. This is a congenital defect similar to an pharyngoesophageal diverticulum. In reality it is a hernia with a definite epithelial sac.

The branchial fistulas occur early in life, and they present themselves as a little dimple low in the neck and anterior to the sternomastoid muscle. They are congenital failure of epithelial closure and consequently burrow down to the lower part of the neck, following the facial planes.

The second arch and pouch develops into the posterior pharynx, the tonsillar fossa, and the tonsil.

The third arch, as it develops along with the pharyngeal pouch, develops into the thymus gland and the lower two parathyroid glands.

The fourth one develops the upper parathyroid glands and contributes to the development of the thyroid.

The fourth cleft forms the lower border of the neck.

[Slide] This slide shows a development of these pouches within the pharynx that grow forward as the arches progress anteriorly to fuse in the midline.

[Slide] There are abnormal developments that take place in the descent of the thyroid gland and that are related to the failure of obliteration of the thyroglossal duct. If there is a failure of obliteration, there is the development of a thyroglossal cyst. The only reason for showing this slide is that so many of these cysts are removed and the midsection of the hyoid bone is not removed along with the thyroglossal cyst and duct. Consequently, there is a recurrence. It is important always to remove the midsection of the hyoid bone in the surgical approach to the problem of thyroglossal cysts.

If the back of the tongue is pushed down by an assistant with a finger in the patient's mouth, there is easier access to the origin of these cysts, which arise from the foramen cecum.

[Slide] This is related to the formation of these abnormal lesions pertaining to the thyroid, such as lingual goiter, intralingual goiter, sublingual goiter, and other abnormalities under the chin and in the midline of the neck such as a dermoid cyst. These are not common lesions but are to be considered.

The pyramidal lobe of the thyroid is a remnant of incomplete descent of the thyroid and is found to the left of the larynx, and it is observed in about 75% of thyroid operations if search is made for it. It should always be removed. If left, it will hypertrophy and become an unsightly mass on the side of the neck. It may also result in a continuance of hyperthyroidism after operation or be a factor in the recurrence of thyrotoxicosis.

Dr. Jackson's paper is very complete and is characteristic of his usual presentations from the

point of view of thoroughness and clinical application.

MR. TOBY LEVITT, Chicago: As Dr. Jackson has covered the subject so inimitably well, I feel there is very little I would add in general, but I would like to draw your attention in particular to one aspect of the problem with reference to the thyroid gland.

[Slide] Any organ, as it ages, has a diminution of epithelial tissue, and in the endocrine glands I have noted an increase of lymphoid tissue with a diminution of epithelial tissue. As the lymphoid tissue diminishes the fibrous tissue increases, so that in the thyroid gland I could trace six arbitrary phases, which could just as well be 600, and in my opinion they are continuous.

[Slide] First, in the thyroid, I found that the epithelial tissue increased; then the lymphoid tissue increased as the epithelial tissue diminished. The lymphoid tissue increased to form a small perivascular local follicle. This resulted by growth in a diffuse lymphoid hyperplasia, and as the process increased I found a diminution of lymphoid tissue and an increase of fibrous tissue with terminal fibrosis. One could name six arbitrary phases.

I could postulate an epithelial hyperplasia simulating that of Graves, a lymphoepithelial hyperplasia resembling that of the Graves constitution, a focal lymphoid hyperplasia with the features of lymphoid thyrotoxicosis, a diffuse lymphoid hyperplasia simulating that of a lymphadenoid goiter, a fibrolymphoid hyperplasia resembling that of Hashimoto, and a fibrosis simulating the first of the three cases of Riedel.

In a study of over 2000 consecutive thyroidectomies I could trace a transition of one phase leading into the other, and the number of patients diminished gradually and evenly with the six phases.

[Slide] I found that in studying these six phases clinically, macroscopically, and microscopically, the patient aged as these phases advanced. The toxicity diminished; exophthalmos firstly increased and then diminished; preoperative loss in weight increased; postoperative hypothyroidism gain in weight increased, and hypothyroidism increased in this series.

The macroscopic study showed that the size of the gland increased and subsequently diminished; the surface of the gland became more nodular and eventually became tied down to the surrounding structures; the capsule became thicker; the vascularity diminished, and the pressure symptoms increased.

It may be practical to appreciate how this concept affects our practical treatment, first, when we see the patient in the clinic or the office, and, second, when we see the patient in the operating room. That is the point I want to make this morning.

This shows the six phases. In my opinion the first and the second phase could on occasion be suited for thiouracil treatment of the patient who has a slightly toxic flare of puberty, pregnancy, or parturition and who has to be prepared for operation with subsequent use of strong iodine solution a week before thyroidectomy. Radioiodine performs a useful function in the first phase.

In my opinion, partial thyroidectomy is still the ideal operation. It is important in the more advanced phases, when the gland has become nodular, when we cannot make up our mind whether the condition is malignant, and when we know there are pressure phenomena so that we are able to inspect and palpate the gland to exclude any malignant change. We frequently have to operate to diminish pressure phenomena.

As the thyroid phases progress, the need for ligation of the pedicles becomes less and that of retaining a bigger residue at operation becomes greater.

In a similar manner, the necessity for giving thyroid extract after operation increases as the phases progress.

DR. ARNOLD JACKSON, Madison. To paraphrase that late popular song, "Give me five minutes more", I am sure we could spend the entire morning discussing this subject.

I do want to thank Dr. Knepper, Dr. Hendrick, Dr. Hunt, and Mr. Levitt for adding their interesting comments to this discussion. I can readily see that I will have to revise my classification right away and add this case of desmoid and give it a little Anglo-American touch at the finish in honor of Mr. Toby Levitt.

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Retromandibular Parotid Tumors

Their Surgical Treatment and Mode of Origin

H. MASON MORFIT, M.D., Denver

One thinks of the parotid gland as lying external or superficial to the ascending ramus of the mandible. About 80% to 85% of the parotid does in fact lie in such a position. There is a small portion, however, which hooks around the posterior margin of the mandible and extends for variable distances medial to this structure. Tumors arising from this retromandibular portion present an entirely different problem, from the point of view of both symptoms and management, from that which is encountered in the classic and better-known tumor arising from the superficial portion of the gland.

Judged from the clinical histories of patients with this tumor, their therapy has been the source of considerable perplexity on the part of the physicians to whom they have applied for help. Their occurence in such an anatomically inaccessible area has led some to declare such growths as "inoperable" or to recommend x-ray therapy as the method of treatment. However, the histology of this group of neoplasms differs in no way from that encountered in other salivary gland tumors, and, since they do not respond well to radiation therapy, surgical removal has come to be accepted as the most effective treatment. This report is an analysis of 12 patients with this disease. A method of surgical exposure and removal is outlined that seems to have been highly successful and is associated with a zero mortality rate and a low morbidity.

From the Department of Surgery and the Bonfils Tumor Clinic, University of Colorado School of Medicine.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 4, 1954.

AGE AND SEX INCIDENCE

Although most tumors of the head and neck have their peak incidence in the middle and latter decades of life, tumors arising from the salivary glands have always defied generalization in this regard. The group of tumors under discussion here is no exception to this statement. Of 12 patients, the youngest was a 7-year-old white boy and the oldest was a 76-year-old woman. The remainder of the patients were scattered rather indiscriminately throughout the decades, and so, apparently, these lesions may be expected to occur at any age.

In any sizable series of salivary gland tumors there is an increased incidence of females over males. This ratio is approximately 60:40. Although our own series is too small to be of any significance, this ratio was not borne out, and in only 2 cases of the 12 were the patients females.

SYMPTOMS, MORBID ANATOMY, AND CLINICAL COURSE

When tumors arise in the superficial portion of the parotid gland, they present the classic preauricular or infra-auricular "lump," usually entirely painless and of relatively slow rate of growth. In the retromandibular portion of the gland, a different clinical picture ensues (Fig. 1). The hard bony barrier constituted by the ascending ramus of the mandible prevents any lateral expansion of the tumor, and so no evidence of growth appears externally. The tumor expands in the only direction left to it, namely medially, and produces a bulge in the lateral pharyngeal wall. This pharyngeal bulge is probably detectable relatively early, but, inasmuch as the lesion at this stage is

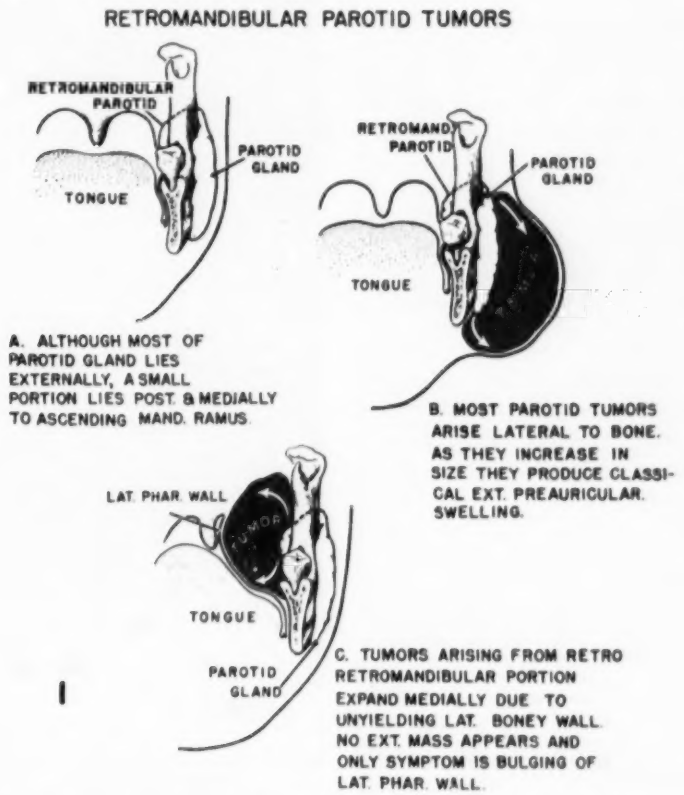

RETROMANDIBULAR PAROTID TUMORS
RETROMANDIBULAR PAROTID
PAROTID GLAND
TONGUE
A. ALTHOUGH MOST OF PAROTID GLAND LIES EXTERNALLY, A SMALL PORTION LIES POST. & MEDIALLY TO ASCENDING MAND. RAMUS.
RETROMAND. PAROTID
PAROTID GLAND
TONGUE
B. MOST PAROTID TUMORS ARISE LATERAL TO BONE. AS THEY INCREASE IN SIZE THEY PRODUCE CLASSICAL EXT. PREAURICULAR SWELLING.
LAT. PHAR. WALL
TUMOR
TONGUE
PAROTID GLAND
C. TUMORS ARISING FROM RETRO RETROMANDIBULAR PORTION EXPAND MEDIALLY DUE TO UNYIELDING LAT. BONEY WALL. NO EXT. MASS APPEARS AND ONLY SYMPTOM IS BULGING OF LAT. PHAR. WALL.

Fig. 1.—Intraoral views in (*A*) Case 2 and (*B*) Case 6 illustrate typical appearance of retromandibular parotid tumors. The classic bulge of the lateral pharyngeal wall due to extensive pressure is well illustrated.

entirely asymptomatic, it is not likely to be discovered at this time.

As the growth increases in size, the bulge in the pharyngeal wall towards the median line becomes more pronounced, and the patient begins to complain of "something sticking in my throat" or a feeling of "pressure" and of having to labor a bit in order to swallow. Even in the advanced stages there is no ulceration of the lateral pharyngeal wall and the bulge can clearly be identified as one due to extrinsic pressure (Fig. 2). If definitive therapy is not instituted, continued increase in size can be anticipated. Eventually interference with the airway and dysphagia will result. Also, as this increase in size occurs, there is extension inferiorly so that the lower pole of the tumor can occasionally be felt presenting below the horizontal ramus of the mandible.

There was nothing in the clinical examination of our patients that permitted us accurately to estimate preoperatively whether a given tumor was benign or malignant. Despite the fact that both of the malignant tumors encountered in this series were firmly affixed to and seemed to be infiltrating the base of the skull, there was no cranial nerve paralysis at the time when the patients were first seen. In both instances, the first hint that we were dealing with a malignant tumor came only after the mass had been surgically exposed and removal was being attempted.

As is true in the superficial parotid tumors, the patients reported considerable variation as to the duration of the lesion.

With the exception of the case of a tumor developing in a 7-year-old boy, which ultimately proved to be malignant, the histories of the remainder of the patients indicated a slow rate of growth. In the case of the 76-year-old woman the growth had been present for at least 30 years. Although this particular lesion was apparently not a very aggressive one, even in this instance difficulties with airway of sufficient severity to threaten death resulted from its continued increase in size.

DIFFERENTIAL DIAGNOSIS

The majority of intraoral neoplasms arise from the mucosa lining the mouth or pharynx and on clinical examination clearly present as surface lesions, usually with ulceration. This group of lesions can immediately be excluded by that fact that the mucosa apparently is not involved in these retromandibular parotid lesions but is smooth throughout, only its contour being changed by the extrinsic pressure. In addition to the major salivary glands (parotid and submaxillary), the oral, nasal, and pharyngeal cavities are lined with small mucous secreting glands (minor salivary glands) which occasionaly give rise to tumors. The histologic picture of minor salivary gland tumors is similar to that seen in the lesions arising from the major salivary glands, and distinction must be based on clinical examination alone and in particular on the anatomic site of origin. Such minor salivary gland tumors can conceivably provide difficulty in differentiation, but it is to be recalled that these tumors occur indiscriminately throughout the entire oral cavity. The retromandibular parotid tumors always present as a bulge in the posterior lateral pharyngeal wall and as such should be easily distinguished from the above.

Other possibilities exist which may produce a similar clinical picture, but these are even rarer than the parotid tumors arising in this area. For example, an aneurysm of the internal carotid artery will produce a bulge in the lateral pharyngeal wall. However, the characteristic expansile pulsation will aid in its recognition. Certain advanced cases of carotid body tumors will also produce a pharygeal bulge, but only after a considerable body of the tumor mass is present also in the neck. Chordomas may cause some confusion, but in this instance the lesion arises characteristically in the midline and produces its main distortion of the posterior pharyngeal wall rather than of the lateral walls. Lymphosarcoma primary in the tonsil can be usually clearly identified as arising in this structure and distinguished from a growth of extrinsic origin producing pharyngeal distortion. Sarcomas of soft tissue origin can conceivably cause difficulty in diagnosis, but to date this has not been a problem. In the event of any doubt, a biopsy will settle the issue. In only one case has this been necessary. Retropharyngeal abcesses can be distinguished by pain and their toxic course.

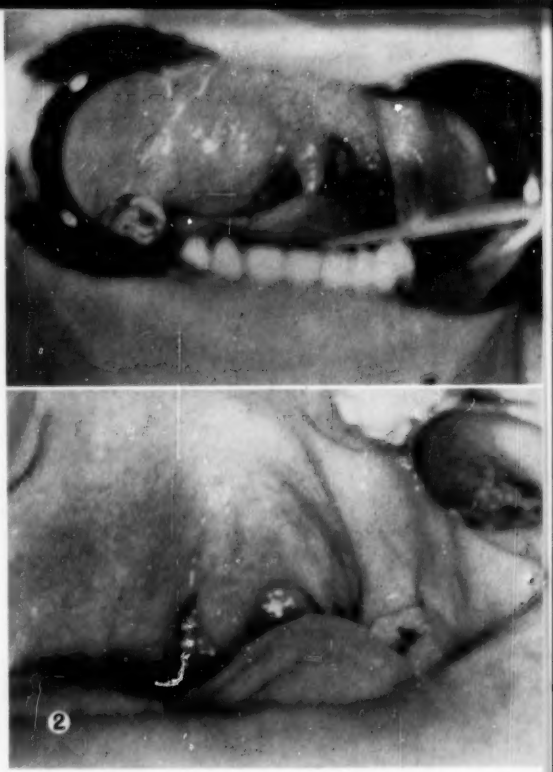


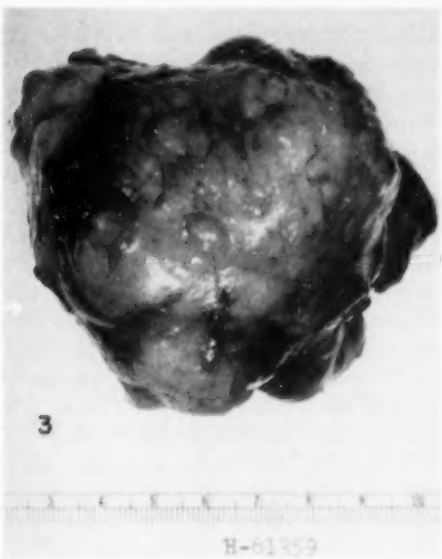
Fig. 2.—Photograph of specimen removed in Case 2. This tumor had been present for 30 years and had slowly increased in size. It measured 8×9 cm. after removal.

OPERATIVE TREATMENT

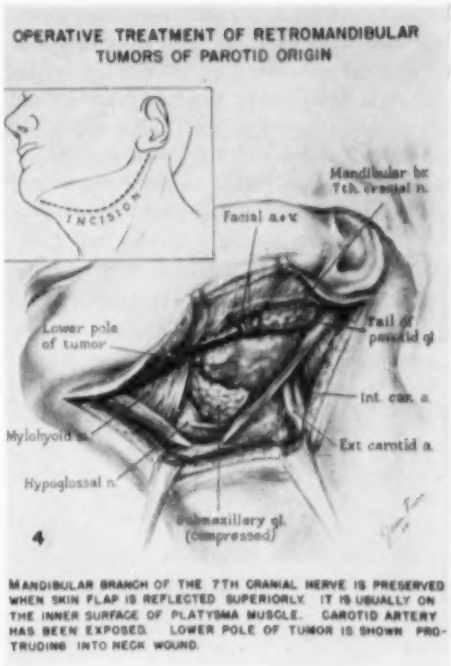
Because the presenting symptoms are intraoral, these patients are likely to be first

sent to an otolaryngologist for definitive treatment. Such physicians are primarily trained in schemes of removal involving intaroral approaches. Removal through such an approach would be a very formidable if not impossible procedure. Indeed, the size of these tumors (Fig. 3) is such as to make it completely impossible to deliver them even through the widest of mouths, assuming that other technical problems, such as maintenance of airway and control of bleeding, were surmounted.

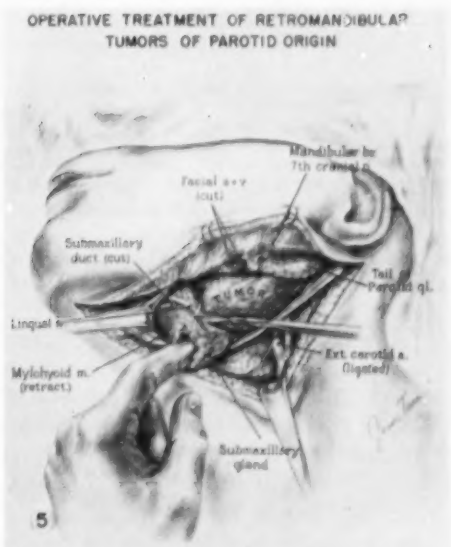
The most successful approach lies in an incision made through the neck with ex-



posure of the pharyngomaxillary space through the submaxillary triangle (Fig. 4). An incision is made through the skin, subcutaneous tissue and platysma muscle extending from the midline anteriorly and curving posteriorly to the level of the tip of the hyoid bone. This flap is mobilized in a superior direction, with preservation of the lowermost branch of the seventh nerve. The facial artery and vein are identified and divided between ligatures as they cross the mandible. The submaxillary salivary gland must be sacrificed in order to gain adequate



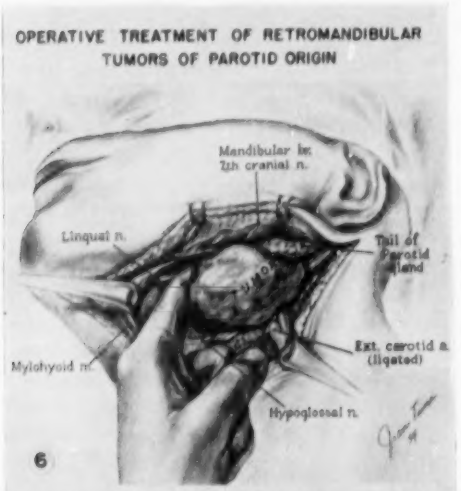
MANDIBULAR BRANCH OF THE 7TH CRANIAL NERVE IS PRESERVED WHEN SKIN FLAP IS REFLECTED SUPERIORLY. IT IS USUALLY ON THE INNER SURFACE OF PLATYSMA MUSCLE. CAROTID ARTERY HAS BEEN EXPOSED. LOWER POLE OF TUMOR IS SHOWN PROTRUDING INTO NECK WOUND.



FACIAL ARTERY AND VEIN ARE CUT AND LIGATED. MYLOHYOID MUSCLE IS RETRACTED. WHEN INFERIOR TRACTION IS PLACED ON THE SUBMAXILLARY GLAND THE NERVE FROM THE LINGUAL N. TO THE SUBMAXILLARY GANGLION IS READILY IDENTIFIED. THESE ARE CUT AND A WAY IS OPENED TO THE PHARYNGO-MAXILLARY SPACE WHERE BULK OF TUMOR IS LOCATED.

exposure. This is accomplished by retracting the mylohyloid muscle anteriorly and pulling the submaxillary salivary gland inferiorly. When this is done, the lingual nerve will come into view (Fig. 5). The autonomic fibers running from the lingual nerve into the substance of the submaxillary gland are identified and divided. The lingual nerve will then spring back up under the mandible out of the way.

The submaxillary duct is identified and divided. The hypoglossal nerve traverses



EXT. CAROTID A. IS LIGATED TO CUT DOWN BLEEDING. THEN TUMOR IS BLINDLY ENUCLEATED AND WORKED OUT NECK WOUND THROUGH WHICH FINAL DELIVERY IS ACCOMPLISHED.

the inferior portion of the submaxillary triangle but can be pushed out of the way and offers no difficulty. The bifurcation of the carotid artery is then exposed, and the external carotid artery is tied off to reduce bleeding. The external maxillary artery is identified as it enters the lowermost portion of the submaxillary gland, and this is divided; subsequently the submaxillary gland can be lifted free of the wound.

At the completion of this step, the lowermost pole of the tumor mass either can be visualized protruding below the horizontal ramus of the mandible or, if the tumor is small, it can be felt by inserting a finger up beneath the mandible. The remainder of the procedure consists of a blind enucleation of the tumor, which is accomplished by inserting a finger behind the mandible into the pharyngomaxillary space and seeking a plane of cleavage (Fig. 6). It is at this point that the first accurate appraisal can be made as to whether the tumor is a benign or a malignant one. If it is benign, there should be no difficulty in finding a satisfactory plane of cleavage. On the contrary, if it is malignant, the operator will find himself working in an apparently satisfactory plane of cleavage only to run into an obstruction due to infiltration by the tumor of either the pterygoid muscles, medial side of the mandible, or the base of the skull.

As the enucleation proceeds, the tumor becomes more mobile, and it becomes possible to pull it inferiorly until it is ultimately delivered from beneath the mandible. Care must be taken to insure that the enucleating finger does not hook around the lingual nerve or the internal carotid artery and avulse these structures in the attempts to remove the tumor. There is always a fair amount of bleeding from the raw tumor bed, and this is controlled with a temporary pack. In only one case has bleeding been any problem. In this particular person (Case 2) it was necessary to leave a pack in place for 36 to 48 hours, after which time it was gradually withdrawn. In our first few cases the possibility of edema of the hypopharynx and larynx owing to the manipulation required for delivery of the tumor caused some concern. An elective tracheostomy was therefore done at the conclusion of each of these operations, but it proved not to be necessary and the tube was removed the following day. Some difficulty in swallowing is to be anticipated for several days after the operation, although the patients have been able to resume their regular diet in every case within three to five days postoperatively. No operative deaths have occurred in this series, and the patients have usually been discharged from the hospital within from 4 to 10 days postoperatively.

PROGNOSIS

Classic parotid tumors have always had a reputation for a high recurrence rate after surgical removal. In some hands this rate has been so high that some have taken the stand that, if the patient is followed long enough, a recurrence will develop in every instance. As the natural history of these growths was studied in greater detail, a satisfactory explanation for this high recurrence rate was provided. Grossly, these lesions appear to be enveloped by a well defined capsule. The surgeon, recognizing the fact that he was working in close proximity to the seventh nerve, was likely to depend too much on this barrier capsule and "shell the tumor out" in his desire to remove the tumor without damaging the seventh nerve.

Surgical pathologists have now established the fact that although grossly this capsule may appear to be the limiting barrier to the tumor, in actuality small nests of tumor cells lie within the thickness of the capsule itself. With the improved techniques now available which involve dissection of the seventh nerve and attempted removal of all of the parotid gland, a much wider margin around the tumor is obtained and a corresponding improvement in the recurrence rate has resulted. The herein described scheme for removing these retromandibular tumors admittedly does not achieve any such wide margin. The anatomy in this area does not permit this to be accomplished. Nevertheless, in none of the benign tumors has there been any evidence of recurrence to date. Our oldest patient was operated upon seven years ago, and the most recent case, one year ago.

The capriciousness of salivary gland tumors has long been recognized, and a parallel is often drawn between their behavior and that of tumors arising in the thyroid gland. In one of our patients (Case 2) a large tumor was removed in January, 1948. The operation proceeded uneventfully until the dome of the tumor was approached, in the region of the base of the skull. At this point, fixation and failure to find a good cleavage plane (which we believe to be characteristic of a malignant tumor) was encountered. When the specimen was ultimately delivered, it was apparent that the superior edge was raw and that the tumor had only been delivered at the expense of fracturing it and leaving a portion of it attached to the base of the skull. Histologic examination confirmed the clinical suspicion that this was a malignant tumor. Despite the fact that we recognized incomplete removal, this particular patient has had no further difficulty to date (seven years).

The effectiveness of tumor therapy is usually measured in terms of the number of patients who remain symptom-free after definitive therapy for a five-year period. This has proved to be a practical yardstick in most instances. When it is applied to tumors of salivary gland origin, however, it has distinct limitations. None of our patients has been followed long enough for us to be able to speak authoritatively in terms of truly long-term results. The quick recovery from the operation plus the immediate relief of symptoms and the failure of any of our patients with benign tumors to develop recurrences during the periods that they have been under our observation has encouraged us to believe that if the tumors are removed by the above described technique the prognosis is good.

REPORT OF ILLUSTRATIVE CASES

CASE 1.—History: The patient was a 47-year-old white man who had been aware of a tumor in his throat for at least eight years. He consulted at least three physicians in his own home town, none of whom made any specific recommendations as to therapy. There were no symptoms such as pain, difficulty in swallowing, or dyspnea, but the patient did complain of a sensation of something "sticking in his throat." He was ultimately referred to a large clinic, where he was told that the growth could not be surgically removed and that he should receive irradiation.

A total number of 30 x-ray treatments were administered to the growth, and the patient states that he believes the tumor may have shrunk somewhat in size as a result of this treatment, although there was never a time when he was not able to look into his throat and see a large bulge in his pharyngeal wall. This continued to worry him, and he persisted in his efforts to get help and consulted six different physicians, none of whom recommended any definitive therapy.

Operation: He was operated upon on Jan. 16, 1948. A tumor 5 cm. in diameter was removed from the pharyngomaxillary space by the above-described technique, without any difficulty. A tracheostomy was done but proved unnecessary. The patient was discharged from the hospital on P. O. 7.

During the course of the operation the horizontal ramus of the mandible was divided, since it was felt that the tumor was so large that it could not be delivered unless this was carried out to gain wider exposure. The mandible was wired together again at the conclusion of the operation, and the patient had no further difficulty until approximately four months after the operation. At this time he returned with radiation necrosis of the mandible. A technical error had been made first of all in dividing the mandible (since it has been found unnecessary to do this in any of the other cases in which the tumors have been even larger than this one) and, in the second place, in dividing it in an area which had been previously exposed to ionizing radiation energy. He continued to expel small bits of necrotic bone up to a year afterward, but the condition was stabilized after that time and he has had no further difficulty. He is now living and well after seven years.

Case 2.—History: This patient was 76 years old when she was first seen and gave a history of having had a tumor for at least 30 years. During that period she had been seen by numerous physicians, with no specific recommendations being made for removal of the tumor. Because of the fact that as the tumor progressed it had comprised her airway and made it impossible for her to eat with any comfort, she finally reported once more to the hospital in search of help.

Examination: This tumor was the largest one encountered in our series. It had originated on the left side and had pushed the pharyngeal wall almost completely over to within 1 cm. of meeting the right pharyngeal wall. Extensions of the tumor could be palpated in the upper portions of the neck as it protruded beneath the horizontal ramus of the mandible. Intraoral examination revealed the tongue to be pushed forward so that it protruded from the mouth and there was such a narrow space left for airway that the patient found it impossible to recline at all without choking and was having considerable difficulty eating without aspirating some of the material into the trachea.

Operation: The patient was operated upon on March 22, 1954. The tumor was enucleated without too much difficulty except that after its deliverance there was considerable bleeding from the extensive raw tumor bed. This was controlled by packing. The specimen was the largest removed in our series, and it measured 8×10 cm. (Fig. 3). A tracheostomy was carried out and proved to be a wise step inasmuch as the patient had considerable edema and it was several days before the swelling subsided sufficiently for her to breathe through regular channels. She left the hospital on P. O. 10 and remains free of trouble to date (10 months).

Case 3.—History: The patient was a 7-year-old white boy, whose parents first noted a swelling protruding from beneath the angle of the right mandible of one month's duration. They consulted their pediatrician, who noted a bulge in the right lateral pharyngeal wall and sought a further consultation. The patient was complaining of some difficulty in swallowing, with occasional regurgitation of liquids through the nose.

Examination: The right pharyngeal wall bulged towards the midline, and on palpation the pharyngeal wall slipped smoothly over the tumor mass; bimanual examination revealed the pharyngeal wall bulge and the mass protruding from beneath the right mandible to be in continuity. A diagnosis of a tumor of retromandibular parotid origin was made, and the patient was admitted for operation.

Operation: On Oct. 10, 1954, the usual approach was made. The operation proceeded without difficulty until attempts at enucleation began. At this stage the efforts to find a satisfactory cleavage plane were repeatedly thwarted. It soon became apparent that there was direct extension of the tumor into the pterygoid muscles and the base of the skull, and any effort to find a cleavage plane in these areas always resulted in the finger being poked into the substance of the tumor itself. Before the attempts at removal were completed, the tumor had become badly fragmented, and the discouraging picture of a malignant tumor broken into by the operator and being disseminated widely throughout the wound had developed. It was clear that this lesion had all of the gross characteristics of a malignant one and that satisfactory surgical removal was impossible to achieve. The wound was closed, and attempts are now being made to control the remaining tumor with postoperative radiation therapy.

Case 4.—History: A 28-year-old white man, who complained of a "lump in his throat" of approximately one year's duration, had been seen by several physicians during his term of service with the armed forces, but no specific therapy had been recommended.

Examination: Intraoral examination revealed the classic smooth bulge of the pharyngeal wall owing to extrinsic pressure. Examination of the corresponding side of the neck failed to reveal any direct extension of the tumor into the area.

Operation: The patient was operated on April 17, 1948. The operation proceeded satisfactorily until enucleation of the tumor itself began. This appeared to be progressing satisfactorily so far as mobilization of the lower pole of the tumor was concerned. As the operator reached the dome of the tumor,

7 RETROMANDIBULAR PAROTID TUMORS

COMPILATION OF CASES (NOV. 1954)

CASE	AGE	SEX	HISTORY	PATH. REPORT	OPERATION DATE	CURRENT STATUS
1 (DP)	47	M	LUMP IN THROAT 8 YRS. X-RAY Rx WITHOUT EFFECT	MIXED TUMOR	1-16-48	L.&W. 7 YRS.
2 (E.W)	28	M	"LUMP IN THROAT" 4 YRS.	CARCINOMA	4-17-48	L. & W. 7 YRS.
3 (W.S.)	37	M	DIFFICULTY IN SWALLOWING 8 MOS.	MIXED TUMOR	2-4-49	L. & W. 6 YRS.
4 (H.S.)	44	M	"PRESSURE" IN THROAT 1 YR.	"	9-11-49	L. & W. 5 YRS. 2 MOS.
5 (J.J.)	24	F	DIFFICULTY IN SWALLOWING 4 MOS.	"	10-2-50	L.&W. 4 YRS. 2 MOS.
6 (W.J.)	54	M	"SOMETHING CAUGHT IN THROAT" 6 MOS.	"	11-1-50	L.&W. 4 YRS. 1 MO.
7 (H.P)	38	M	DIFFICULTY IN SWALLOWING 1 YR.	"	4-4-51	L.&W. 3 YRS. 7 MOS.
8 (O.H.)	42	M	MASS DISCOVERED IN ROUTINE PHYSICAL EXAM	"	9-3-52	L.&W. 2 YRS. 2 MOS.
9 (H.E.)	29	M	MASS DISCOVERED IN ROUTINE PHYSICAL EXAM	"	11-4-53	L.&W. 1 YR.
10 (G.T.)	30	M	"SOMETHING PRESSING IN THROAT" 8 MOS.	"	1-15-54	L.&W. 10 MOS.
11 (E.C.)	76	F	TUMOR OF THROAT 30 YRS.	"	3-22-54	L.&W. 8 MOS.
12 (P.R.)	7	M	MASS NOTED BY PARENTS AT ANGLE OF MANDIBLE 1 MO.	ANAPLASTIC CARCINOMA	10-2-54	LIVING WITH DISEASE

however, in the region of the base of the skull, the plane of cleavage was obliterated and difficulty was encountered in securing final delivery. When this was finally achieved, gross examination of the specimen revealed a raw area approximately the size of a nickle presenting the appearance of having been actually fractured away from the base of the skull. The probability that of residual tumor infiltrating this area was being left behind was recognized.

Pathological examination confirmed the gross impression of a malignant tumor, and although postoperative radiation therapy was recommended the patient never returned for this therapy. It had been assumed until this report that the patient had probably died from his disease. A recent follow-up, however, revealed that the patient is still alive and has had no further difficulty of any kind and is busily engaged at his full-time occupation of operating a clothing store.

SUMMARY AND CONCLUSION

A little known and apparently poorly recognized variety of tumor arising from the parotid gland is described. The symptomatology is reviewed, and a practical method of surgical removal is outlined. The absent mortality and low morbidity combined with the fact that no other alternate effective method of therapy is available forces us to look upon this method of surgical removal as the most satisfactory means of dealing with these lesions. The great length of time that must elapse before the word "cure" can be employed in salivary gland tumors is recognized. Our results up to the present time, however, lead us to believe that this is an effective treatment.

Extent of Mandibular Resection Required for Treatment of Oral Cancer

LOUIS T. BYARS, M.D., St. Louis

A properly planned operation for cancer of the mouth often includes the removal of a segment of the mandible or maxilla because oral cancer arises upon these structures or eventually may extend to them. Malignant growths are commoner in the lower than in the upper oral cavity, and the mandible is attacked several times as frequently as the maxilla. The lower jaw is functionally the more important of the two, and resection of it creates a serious defect. A logical plan based upon anatomical and surgical principles and a knowledge of the growth habits of cancer should be used for determining the proper extent of resection without creating needless deformity. This rule should be applied to the individual case rather than treatment being based on routine.

The mandible becomes the host of malignant tumor by one of five methods. It is rarely the site of metastasis. Two instances of metastases from the breast and one from the thyroid gland have been encountered. Equally rare is the formation of primary epidermoid carcinoma within it. The three following channels of direct entry are com-

From the Department of Surgery, Division of Plastic Surgery, Washington University School of Medicine.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 4, 1954.

Fig. 1.—*A*, Drawing of atrophic mandible of type often possessed by cancer patient. Absorption of alveolar process has caused the mandibular canal to become more superficial and accessible for invasion (1). The mental foramen (2) is now submucosal and easily entered by buccal cancer. Channel (3), direct extension of cancer from a cervical node, does not find the mandibular canal much more exposed than is the case in *B*. *B*, Drawing of "conventional" mandible. This structure, containing teeth, is more massive than the atrophic counterpart, *A*. While it may be invaded by the three common routes, the mandibular canal is less exposed and, as a result, the operation of resection and disarticulation is less often necessary.

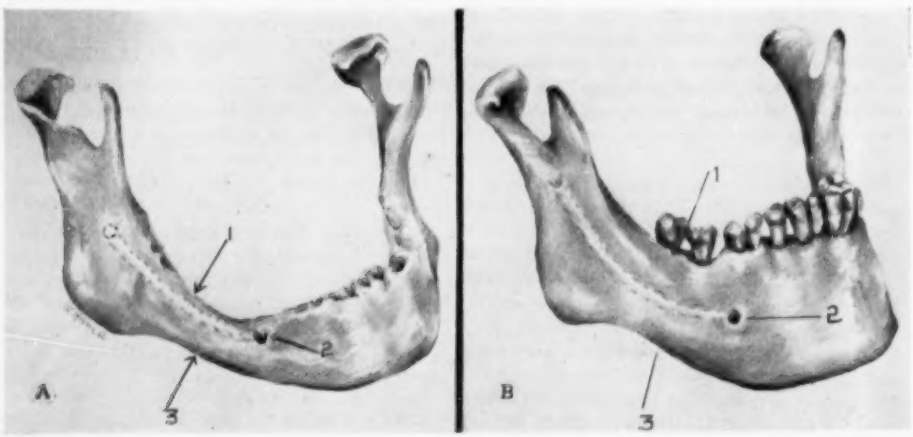
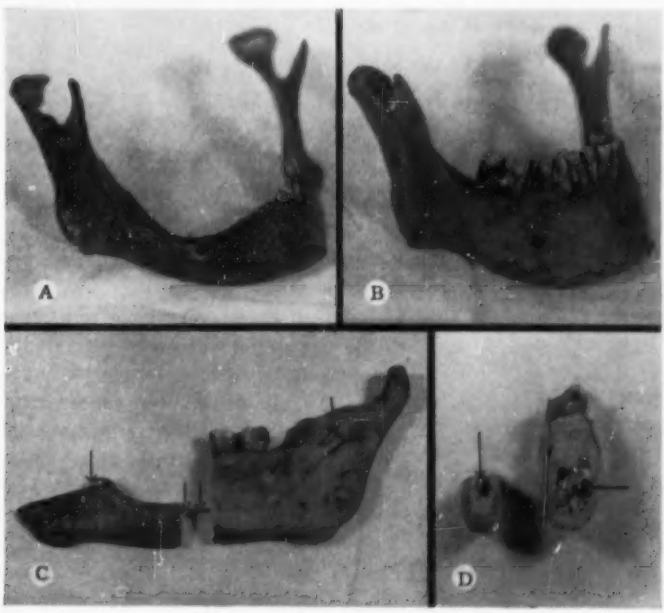


Fig. 2.—X-ray of atrophic mandible eroded by cancer arising on the alveolar process. Marker shows point of penetration into the mandibular canal, which is of shallow depth. Enlarged caliber of canal central to the point of entry indicates progression of cancer along the axis of the mandible. When this canal has been reached by cancer the mandible must be disarticulated. Segmental resection would leave a portion of mandible with cancer in its core.

mon, however (Fig. 1). 1. Most frequently, an advancing primary lesion of oral mucosa penetrates its integument, involves the upper surface, and enters the bone. 2. Invasive cancer of the lip may spread to the region of the mental foramen, enter, and travel up the core of the mandible. This route is also followed by lesions of the oral mucosa in the cuspid region. 3. Another manner is comparable to the first, but the port of entry is the lower mandibular border rather than the upper. Cancer of the lip and sometimes that of the face and cheek metastasizes to the "buccal" lymph node (Fig. 5). This is a node in the cheek-neck position occurring at the point where the facial artery crosses the lower border of the mandible. Such a node is separated from the bone by a bit of areolar tissue, quickly becomes parasitic upon the mandible, and invades it by direct encroachment (Fig. 6).

Bone must be considered to be invaded by oral cancer when a malignant process has penetrated its thin mucosal covering. The alveolar soft tissue is 2 mm. in depth, and even the earliest growth descends to the

Fig. 3.—*A* and *B*, normal intact mandibles illustrating tremendous changes which occur with age and loss of teeth. *C* and *D*, Segments of similar mandibles cut to permit the insertion of soft lead wire through the mandibular canals. These wires are accurately placed at point where inferior dental nerve traversed the bone. As senile atrophy develops the canal is very superficially located in comparison with its original site and is readily invaded by cancer. (Reproduced with permission of *Surgery, Gynecology and Obstetrics*, Chicago, May, 1954, published by the Franklin H. Martin Memorial Foundation.)



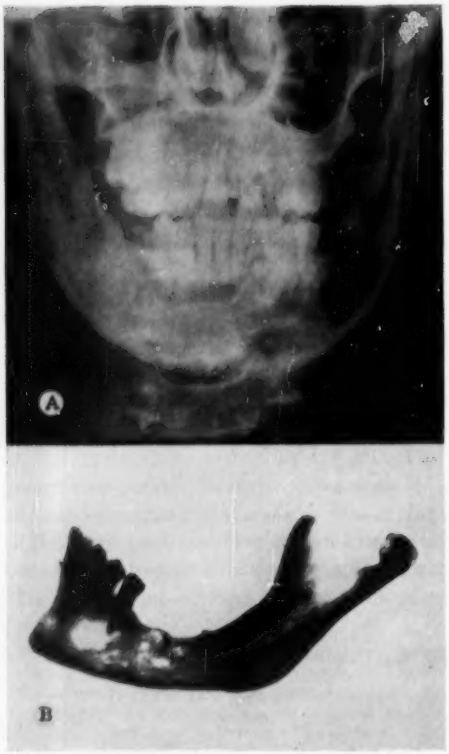
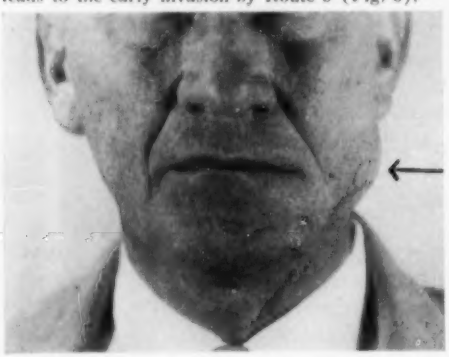


Fig. 4.—*A*, preoperative x-ray of mandible of patient "cured" of cancer of lip only to die eventually from residual tumor which entered the mandible by way of the mental foramen. *B* is film of bone removed at operation and shows greater detail of destruction along mandibular canal. At time of first operation for the lip tumor, the growth extended to the mental foramen and the mandibular invasion should have been suspected. In the atrophic mandible the mental foramen is immediately submucosal and exposed to entry by any malignant lesion of the cuspid region.

Fig. 5.—Cancer of the lip and sometimes other malignant lesions may metastasize to this "cheek-neck" node overlying mandible. Such a condition leads to the early invasion by Route 3 (Fig. 6).



periosteum; the more advanced growth produces gross erosion. Thus in every such case bone excision must be considered.

Bone is a barrier to intraoral cancer spread because of its density. Once tumor has reached bone, the periosteum is invaded and lateral extension develops. This presumption of bone invasion is based upon clinical and microscopic findings and is not revealed by x-ray examination. The next stage, surface erosion, may be seen on a well-prepared dental film, but because of technical difficulties it is often not revealed by the usual jaw roentgenogram. The penetration is greater than is suspected, since visible changes are preceded by less obvious ones.

Fig. 6.—X-ray of mandible invaded by Route 3 (Figs. 1 and 5). If the mandibular canal is approached, resection and disarticulation are indicated.

The direction of cancer growth is always determined in part by natural barriers and paths of least resistance. Usually the intraoral growth has a wider mucosal spread than periosteal penetration. The periosteal area may be still greater than cortical bone erosion. However, the body of the mandible contains a channel, the inferior dental canal,

which again permits unobstructed lateral progress if reached. Thus the surgeon must visualize an "iceberg" situation, in which more is hidden than is revealed when this mandibular canal has been reached by invading cells (Fig. 2). This situation does not hold for more superficial lesions.

The canal transmits lymphatics, blood vessels, and the dental nerve. It begins at the mandibular foramen on the inner surface of the ramus just below the sigmoid notch, transverses the ramus and body of the lower jaw bone, and terminates at the mental foramen on the outer surface near the cuspid region (Fig. 1).

When the mandible is involved, the surgeon may select one of three procedures. The simplest operation is the resection of the cancer along with the periosteum and a plate of underlying bone, not interrupting the continuity of the mandible. This conservative approach must, of course, be reserved for the lesions of minimal extent.

More frequently a radical resection is necessary. A full-thickness segment may be excised, or the mandible may be resected and disarticulated. The simple segmental resection is quicker and easier to perform, is followed by smoother postoperative recovery, causes less deformity, and lends itself to secondary repair if such is indicated. It may also be an inadequate procedure under certain conditions.

The basic consideration in making this decision is whether or not the inferior dental canal with its vessels, nerves, and lymphatics has been entered by cancer cells. It forms a natural path for cancer dissemination and, if invaded, any operation other than adequate jaw resection with disarticulation at the temporomandibular point is likely to leave behind a segment of mandible with cancer growing in its core (Fig. 4).

When selecting the type of operation to perform, the surgeon may overlook the tremendous alteration in the structure of the lower jaw which takes place with increasing age and the loss of teeth. This change in the character of the mandible causes the centrally placed mandibular canal of the normal adult who possesses full dentition to become progressively exposed on the oral surface until it is covered only by 1 or 2 mm. of bone in the completely edentulous elderly patient (Fig. 3).

The canal is about equally distant from the upper and lower borders of the normal tooth-containing mandible. When teeth are lost, the mandible becomes generally smaller but the alveolar process may be completely absorbed. The canal remains almost as far from the inferior margin of the jaw as it was originally but now lies only a millimeter or so from the upper cortical border. Under this condition almost any surface erosion permits malignant tissue to enter the canal and extend rapidly in a central direction. An operation which is adequate to cure a given lesion in one person would be totally inadequate in another (Figs. 2 and 3).

As a consequence of these anatomical changes the mental foramen also shifts position. In the normal adult with full dentition this structure is on the outer surface of the mandible below the cuspid position and about equally distant from the upper and lower mandibular borders. At the termination of senile atrophy it is on the outer superior surface and so exposed that a lower denture causes its wearer pain because it rests upon the emerging mental nerve. Epidermoid carcinoma of the lower labial sulcus immediately enters this opening and progresses up the canal. Recurrent cancer of the lip not infrequently extends to the same location and produces the same result. In either case the mandible must be removed for best chance of cure even though x-ray evidence of bone destruction is absent. Of course, a malignant process of sufficient extent may follow this same course in the normal mandible, but the foramen is more protected (Fig. 4).

Certain lip, facial, or intraoral cancers metastasize first to the upper cervical lymph nodes. These nodes become fixed to the periosteum early and invade the mandible from below, just as intraoral growth does from above. The same principles of treatment hold. Fixation requires bone removal; erosion necessitates segmental resection, and

if there is likelihood of mandibular canal involvement resection and disarticulation are required. When bone erosion is evident on x-ray examination, its actual extent is greater than is shown (Figs. 5 and 6).

OPERATIVE PROCEDURE

All operations in which a full-thickness removal of mandibular bone is contemplated are performed with neck dissection in continuity. Great care must be exercised to prevent the seeding of malignant cells in a receptive wound during the execution of this surgery, because the primary lesion is at the surface and at the same time hidden in the depths of the mouth, where sponging and retraction are likely to break off tumor particles. One safeguard is to obtain good exposure, and in every instance an incision is made from the lip border to the transverse arm of the neck dissection incision so that the entire cheek may be reflected, leaving the primary lesion attached to the mandible and accessible.

The dissection is limited to the digastric triangle in rare instances, usually when the primary growth is of the verrucous type. All too frequently the primary region and upper neck remain free of disease only to have metastases appear in the lower neck when the dissection has been of a partial nature. The operability of a given lesion is not questioned just because bone is invaded or because a large bulk of tumor is present. A decision that a specific malignant process is too advanced to be amenable to surgery should not rest upon the consideration of the immediate postoperative deformity. A large area of soft tissue in the form of mucosa, cheek skin, or the full thickness of the cheek must often be sacrificed in the course of an adequate operation. Such defects can be repaired satisfactorily if the patient is cured. However, much may be done at the time of surgery to minimize discomfort, protect the patient's airway, aid swallowing, and facilitate later reconstruction if pharyngeal collapse is prevented by controlling remaining jaw fragments and holding them in as nearly normal anatomic relationship as possible. Proper attention to mandibular support usually obviates necessity for tracheostomy.

PRESERVATION OF MANDIBULAR SUPPORT

As soon as the resected tissue has been detached and bleeding has been controlled, mandibular continuity and support are secured. Usually skin flaps have been sutured to cover the lower neck wound in the interest of asepsis before the mouth is opened.

If a simple segmental resection of the mandible has been done, the procedure is simple. The gap is bridged by the insertion of a bar of Stainless Steel of appropriate length between the bone ends. The densest bone is drilled, and the bar is inserted snugly into the drill holes in the ends of the bone. If this bar is inserted so that it is straight and in line with the mandible, it will continue to penetrate the bone and eventually telescope. In the average case the resected segment is long enough that ramus must be united to the symphysis or a point near it. This procedure permits the bar to be bent at the ends and inserted so that the portions contained within the bone are not in a

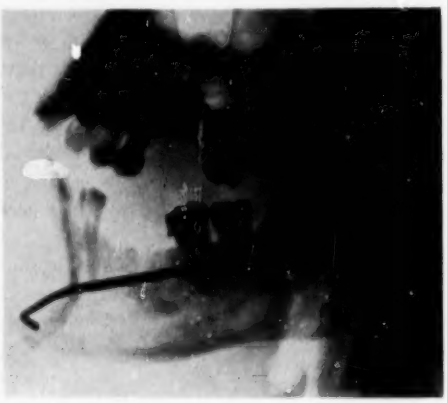


Fig. 7.—Prosthesis of Stainless Steel inserted at time of segmental resection to maintain position of mandibular fragments and to support oral and pharyngeal structures. This is usually a temporary splint to minimize postoperative complications but is occasionally permanent. (Reproduced with permission of *Surgery, Gynecology and Obstetrics*, Chicago, May, 1954, published by The Franklin H. Martin Memorial Foundation.)

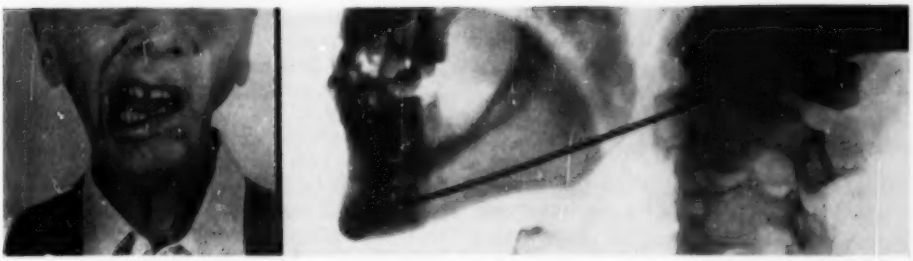


Fig. 8.—Patient with temporary internal bar splint in position three weeks after resection and disarticulation of the left mandible. Prosthesis is threaded Kirschner wire drilled into mastoid tip behind and inserted into symphysis in front. This supports the jaw, mouth, and pharynx during the postoperative period and makes for an easier recovery.

straight line with the part bridging the gap between bone ends. Thus, the stress is distributed so the metal ends stop boring through the rather soft bone (Fig. 7).

This method of fixation is simple, and the prosthesis is easily tailored to fit. No effort is made to shape the bar to conform to the normal curvatures of the mandible. Rather, the prosthesis follows the straightest line between the two points and is so placed that it will lie between inner and outer soft tissue covering without pressure on either. Soft tissue covering such a metal prosthesis under tension will usually be cut through eventually. As secure a closure of intraoral tissue is made as possible. The overlying skin is closed with dependent drainage.

The material used should be of inert metal which can be bent and cut easily with ordinary wire pliers and cutters at the operating table. As its function is to maintain the distance between the bone ends and to prevent uncontrolled motion, it must be of adequate caliber and rigidity. Certain disadvantages arise from the use of too great thickness. A suitable size of Kirschner wire can serve as the prosthesis.

In a few instances following cancer operations the internal support has healed in place and several months later a bone graft has been inserted to bridge the space preserved by the prosthesis. No splint or appliance other than the bar has been necessary for fixation. A deficiency of soft tissue to house the graft may prevent this type of repair (Fig 9).

If a resection of the mandible with disarticulation at the temporomandibular joint has been done, the problem of internal splinting is more difficult. If it is believed that the

Fig. 9.—*A*, in favorable cases the internal splint may heal in position and be supplemented with a bone graft later. The splint is inserted as a straight connection between the cut bone ends rather than with any attempt to shape it like the resected bone. The bone graft is then placed so as to reproduce the mandible and is not in contact with the metal bar. This x-ray shows the bone graft six years after jaw resection. The bar is still in place. The bar is superior to the graft but not as distant from the graft as the distorted picture would suggest. *B*, photograph taken five and one-half years after bone graft, six years after jaw resection.

splinting is definitely to be temporary, a threaded Kirschner wire is used. One end is drilled into the mastoid process posteriorly. The threads grip the bone and prevent slipping. The anterior end is inserted snugly into a hole drilled in the cut end of the mandible. Such a prosthesis gives good mandibular support, permits mouth opening because of its flexibility, and is tolerated comfortably (Fig. 8). No such support has been left in longer than six weeks because of fear that it might progressively penetrate into deeper, dangerous areas.

FATE OF THE PROSTHESIS

The purpose of the internal support is to make the patient's postoperative course as easy as possible by enabling him to eat and breathe more easily and to reduce pain from movement of bone fragments. It also holds the bone ends apart during the healing period so that the final displacement is less. In most instances it is a temporary splint which is removed after two to six weeks. In some cases the metal bar has become permanent. The degree of permanence depends largely upon the conditions present at the end of the operation.

After completion of an adequate cancer operation sometimes so much skin or mucous membrane has been removed that the metal bar is exposed on the inside of the mouth, on its outer surface, or both. Here its removal is simple after the purpose has been achieved. The bar is cut in the middle and the two ends withdrawn. Under such conditions the prosthesis could not become permanent.

Often intraoral covering can be secured by elevating the floor of the mouth and sufficient skin is present. Frequently the bar will heal in and function perhaps for months until it gradually works loose. It can be exposed through a small incision, divided, and withdrawn. It is desirable that the caliber and temper of the bar used be such that it can be cut with a simple pointed wire cutter; otherwise a more cumbersome compound instrument must be used and a larger incision made to permit its introduction.

DISCUSSION

ON PAPERS BY DRS. MORFIT AND BYARS

DR. DAVID ROBINSON, Kansas City, Kan.: Mr. President and gentlemen, a word first about Dr. Morfit's paper.

I wish I had heard him read this paper a year ago, and I would have saved myself and a patient quite a lot of trouble. First of all, it is remarkable that he has had 12 patients of this kind in his experience, because this is quite a number of this type of tumor for one person to have seen.

The man I am referring to was in his sixties. He came in with a mass presenting in his soft palate. He had come for another purpose. I inadvertently found this mass and told him it should be taken out, thinking it was a mixed tumor of the palate.

I proceeded to take it out through the mouth. I soon encountered the condition of dystocia that Dr. Morfit mentioned, and I wished I had had a Piper aftercoming head forceps to get it out. A finger down deep in the hole around an internal carotid is not a pleasant thing, dissecting blindly. I finally delivered the tumor but left the man with paralysis of the soft palate. He has quite a nasal voice now.

I have seen two or three others of these tumors, not presenting so remarkably as those in continuity with the larger parotid tumor that would be approached from the neck anyway. I think Dr. Morfit's approach is a good one, and I will use it the next time I see such a tumor.

A comment for a moment on Dr. Byars' paper: Largely, the trouble I have had after a jaw resection in instances such as Dr. Byars has mentioned has been in not resecting enough of the mandible. I have a patient now in whom I resected the mandible in a limited sort of way. I found that on cross-cutting through the ramus a cheesy material could be expressed from the inferior alveolar dental nerve. I went back and took out the mandible later, but this man is now dying of his extension metastases.

DR. ROBERT A. WISE, Portland, Ore.: I think we should be indebted to Dr. Morfit for bringing to our attention this very ingenious approach for what in our experience is a rare type of tumor.

We have had four benign tumors occurring in the retromandibular or deep lobe of the parotid gland in a series of about 50 benign tumors. Of these four, two involved the deep lobe alone, while the other two involved not only the retromandibular portion of the parotid gland but also the superficial portion, extending from one lobe of the gland to the other.

The approach we use for these tumors is the one we follow for all benign tumors of the parotid gland. The superficial lobe of the gland is exposed and removed. The facial nerve is isolated and retracted, and the deep lobe is then excised, the facial

nerve being visualized and protected during the removal of the deep lobe.

[Slide] I would like to show an example of a benign tumor involving the deep lobe alone and also one involving the deep and superficial lobes. The first is a very interesting tumor, identical to that described by Dr. Morfit. This is the base of the tongue, the tonsillar pillar, and the tumor presenting in the posterior pharynx. It was removed in the manner described above and was shown to be a mixed tumor.

[Slide] Here is the tumor removed from this deep portion of the parotid gland.

[Slide] Here is an example of a recurrent mixed tumor involving not only the deep lobe of the gland, as seen here, but also the superficial portion, extending in continuity from the deep lobe to the superficial lobe. A total parotidectomy with preservation of the facial nerve was performed.

I would like to ask Dr. Morfit how he handles such a situation with his incision and what protection he can assure the facial nerve during his procedure.

DR. JAMES W. HENDRICK, San Antonio, Texas: The paper by Dr. Morfit on parotid tumors arising from the retromandibular lobe is the first that I am acquainted with that deals with this problem alone.

In the early 1930's while I was visiting in London and attending a clinic conducted by Dr. Hamilton Bailey on parotid tumors, he demonstrated two examples of tumors of the parotid gland that were located in the retromandibular lobe. Dr. Bailey discussed the anatomy of the parotid gland and showed illustrations demonstrating that the parotid gland was dumb-bell in shape, with a large superficial lobe, a small isthmus extending around the angle of the mandible, and a smaller retromandibular lobe. That was my first acquaintance with the retromandibular lobe. When I returned to this country, I had occasion to dissect some 75 cadavers with special reference to the parotid gland and confirmed Dr. Bailey's observation that there definitely was a retromandibular lobe. I wrote a short paper on the anatomy of the parotid gland demonstrating the two lobes, a larger superficial and a smaller retromandibular with the facial nerve beneath the superficial lobe. Also in most instances tumors involving the superficial lobe could be dissected out without injury to the facial nerves. I received considerable correspondence from surgeons over the country relative to the retromandibular lobe, some with adverse comments that no such lobe existed.

Dr. Grant Ward and I studied the salivary tissue tumors at the John Hopkin's Hospital and found that there were eight or nine cases in which the tumor was located in the retromandibular lobe. I have used a technique similar to that advocated by Dr. Morfit for their removal except when primary malignancy is found, in which I include a radical neck operation.

Relative to Dr. Byar's excellent and timely paper, for many years we surgeons that are doing radical head and neck surgery for benign and malignant disease frequently find it difficult to impress on our colleagues that it is essential to resect small or, in some instances, large segments of the mandible. My comments will be from that standpoint.

In a study of carcinoma of the lip, we found that 3% of malignant lesions of the lip that occurred in the lateral third of the lip that were over 2 or 3 cm. in diameter had metastases to the mandible in addition; frequently this was present before there were clinical manifestations of metastases in the lymph nodes of the neck. In a review of lymphatics of the lip you can readily observe, from this slide [Slide], that some of the lymphatic trunks from the lateral third of the lip enter the mental foramen. When we have a patient with carcinoma of the lateral third of the lip over 1 or 2 cm. in diameter, we place one finger in the mouth and one on the outside to give the mandible a good squeeze. If there is involvement, the patient will complain of pain; if there is any evidence of involvement, it is well to have an x-ray film made of that area of the mandible.

In the presence of adamantinoma of the mandible, we have found that it is essential that the full thickness of the mandibie and a good margin on each side of the tumor be resected to prevent a recurrence. We have been unable to eradicate or prevent a recurrence of adamantinoma by curetting the tumor. Approximately 25% of the patients with this disease give a history of repeated curettements of the mandible or maxilla which are always followed by a recurrence.

In some instances, it is necessary to resect as much as 60% or 70% of the mandible, as is illustrated in this slide of a Negro woman who had a huge adamantinoma involving the entire right half of the mandible with the exception of the ascending ramus; the mandible was resected and the graft of rib, as you will note, was inserted at the time of operation. This can be safely done at that time if the oral cavity is not opened during the primary operation. This affords good cosmetic and functional results.

In carcinoma of the tongue and the floor of the mouth, when it is necessary to do a composite operation in which a radical neck resection is also done, a segment of the mandible is resected along with the floor of the mouth and a hemiglossectomy. Various types of prosthesis, as illustrated in these slides, may be utilized. As you will note from some of these slides, the prosthesis with the bar carries teeth, which adds to the cosmetic and functional results. Other types of prosthesis are utilized to prevent the unresected area of mandible from being

pulled to the opposite side, as demonstrated in this slide. Just before leaving San Antonio to attend this meeting, I resected an area of the right mandible in a patient who had been treated elsewhere for carcinoma of the floor of the mouth with radium needles. The primary tumor was small and the cancer was eradicated but there was a resultant oral fistula which permitted a constant drooling of the saliva and liquid food from the right side of the mouth, resulting in marked weight loss and disability to the patient. It was impossible to close the fistula without resecting an area of the mandible which had been reduced to one third of its original thickness by the effects of radiation. After resection of the area of mandible on this patient, his nutrition became better; he had a different outlook on life and could once again enjoy better health.

Dr. Myers has made a careful study of the facial bones in the various age groups, with special reference to the mandible.

Dr. H. Mason Morfit, Denver: I would like to try to answer Dr. Wise's question concerning the facial nerve.

As far as the pure variety (such as we have discussed today) of retromandibular tumor presenting in the pharynx is concerned, with this approach the facial nerve is not a problem. When one is dealing with the larger tumors, such as the second one that he showed, where there is not only pharyngeal extension but a large lateral mass, obviously the problem is considerably more complex.

The only thing I would suggest is to follow the old rule that we try to follow in surgery everywhere in the body, namely, that when we are working in close proximity to an important structure we expose the structure, and with it so exposed we are not as likely to damage it as if we had neglected to expose it.

Therefore, in the larger tumors an attempt probably should be made to expose the seventh nerve. Whether one can save it or not will depend entirely on the conditions that prevail as far as the extent of the tumor is concerned, and in some cases, of course, there is no choice and one simply has to sacrifice it.

Carcinoma of the Thyroid and Other Diseases of the Thyroid in Identical Twins

DAVID W. ROBINSON, M.D.
and
THOMAS G. ORR, M.D., Kansas City, Kan.

Any disease occurring in one identical twin may be expected to occur in the other at the same time or at some future time. In a recent report (1953) of sympathicoblastomas occurring in 3-month-old identical twins Lee[1] stated that "when a tumor, especially a malignant tumor, is recognized in one of identical twins, there is a considerable possibility that a malignant lesion, usually, but not always, of a similar type will appear in the other twin."

In the study of disease in twins one of the difficult problems is the identification of identical (monozygotic) twins. Unidentical (dizygotic) twins of the same sex may so closely resemble each other that they may not be distinguished from identical twins unless one placenta for both is recorded at birth. Rosanoff and associates[2] emphasize this problem, as shown in Table 1, taken from their report of a study of schizophrenia in 142 pairs of twins. In the probably monozygotic group both twins were affected almost three times as often as the probably dizygotic group, and one twin only was affected more than three times as frequently as in the dizygotic group. From this study it appears probable that the same-sex probably dizygotic group were actually dizygotic twins, although they may have had the appearance of identical twins.

The study made by Macklin[3] indicates that approximately the same ratio of incidence occurs for tumors as for mental disease in twins. In Macklin's series of collected cases from various sources both twins were affected in a higher percentage of cases in the monozygotic than in the dizygotic group (66.6% and 20%, respectively). Monozygotic twins were affected at more nearly the same age and more often with the same type of tumor than were dizygotic twins. This author stated that "for the most part, if one waits long enough in the case of monozygotic twins before making the report, the second twin is liable to become affected." After a careful study of the cases recorded in the literature, McFarland and Meade[4] concluded that the following generalization seemed justified:

From the Department of Surgery, University of Kansas School of Medicine.

Read at the Sixty-Second Annual Meeting of Western Surgical Association, Colorado Springs, Colorado, Dec. 4, 1954.

TABLE 1.—*New Cases of Schizophrenia in Twins* *

	Same-Sex Twins				Opposite-Sex Twins (Dizygotic)	Totals
	Probably Monozygotic		Probably Dizygotic			
	Males	Females	Males	Females		
Both affected	10	18	3	7	5	43
One affected	9	4	8	25	Males 21 Females 22	99
Totals	19	22	11	42	48	142

* Taken from Rosanoff.[2] **The American Journal of Psychiatry**, Baltimore, The American Psychiatric Association.

Fig. 1.—Identical twins 24 years of age. Both had papillary carcinoma of the thyroid at the same time.

"Heterologous twins never suffer from identical malformation, nor does one of homologous twins ever suffer from malformations not shared by the other."

It is beyond the scope of this report to attempt an analysis of studies related to the influence of heredity and environment upon like diseases in twins. Such studies indicate that much is still to be learned concerning this problem.* Although some evidence in favor of heredity has been presented, McFarland [7] has stated that before the influence of heredity upon the development of disease in twins can be proved our knowledge of twins will have to be greatly amplified. Approximately 25% of twins are of the identical (monozygotic) type. It is now well established that unidentical (dizygotic) twins are no more subject to like diseases than are ordinary siblings except insofar as heredity may play a role in each. Therefore, the study of unidentical twins would add nothing to our knowledge of the incidence of disease in identical twins or the bearing which heredity may have upon the development of like disease in such twins.

* References 5 and 6.

CASES REPORTED AS TWINS

TWINS 1.—D. H., a 24-year-old nurse at the University of Kansas Medical Center, had noted for two months a nodule in her right midneck. The discovery of another nodule prompted her to seek the advice of her doctor who referred her to the Surgical Clinic for a lymph node biopsy. The node was taken from the right midneck deep jugular chain and contained metastatic carcinoma, probably originating in the thyroid. On the day of her admission, March 23, 1953, her identical twin, D. G., asked to be examined for a similar mass in the right neck. The twin was admitted, then, at the same time as her sister.

On the following day D. H. was operated upon by Dr. Orr and Dr. Robinson. She had total thyroidectomy and a radical neck dissection on the right side. The right lobe contained two hard nodules, and a small nodule 1 mm. in diameter was found in the left lobe. A parathyroid was identified on the left and was not disturbed. The girl's convalesence was uncomplicated, but she had a positive Chvostek's sign for about a week and some tingling of the fingers. Pathologic diagnosis was papillary adenocarcinoma of the thyroid with lymph node metastasis in 11 of the 19 nodes found in the radical neck dissection specimen.

Her twin sister, D. G., was operated upon two days later. Findings at operation, which was done identically as for the first girl, were two nodules in the right lobe with questionable adherence to the recurrent laryngeal nerve. This nerve was

Fig. 2.—The same twins as in Figure 1, showing scars of right radical neck dissections.

dissected free. Examination of the removed specimen showed papillary adenocarcinoma of the thyroid with one lymph node metastasis present in 11 nodes found in the radical neck dissection specimen. This girl did not show any lowered blood calcium or the clinical findings thereof.

Radioactive iodine studies for both girls postoperatively have shown no uptake. Both have delivered normal full-term infants within the past year. There has been no evidence of recurrence. Both girls have continued on a maintenance dose of thyroid daily.

TWINS 2.—Thirty-year-old identical white twin men were admitted to the University of Kansas Hospitals Nov. 8, 1938, and discharged Nov. 30, 1938 (Fig. 4). Their complaints were goiters. One stated that a growth in his neck had been present three or four years, and the other, seven or eight years. There had been a gradual increase in the size of the goiters. The goiter appeared first in the midline in each case. Both had some dyspnea on exertion. In childhood the twins were diagnosed as cretins and given thyroid extract. One was right-handed and the other left-handed.

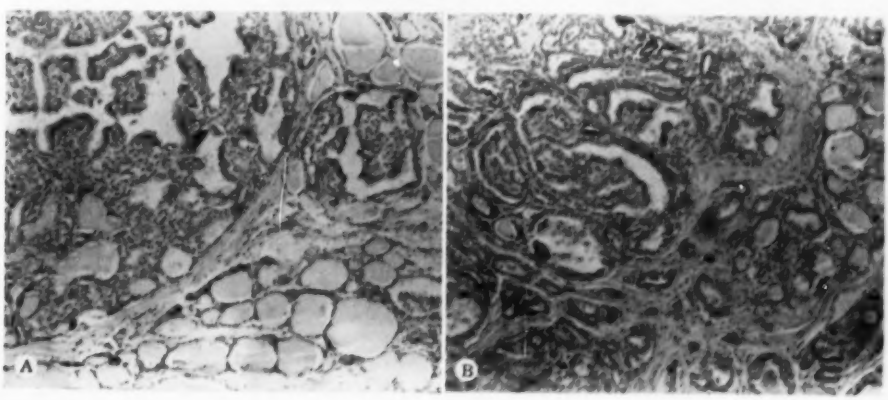
Physical Examination.—Both patients were undersized. They had cretin facies, with dry skin, thick lips, and thick tongues. Both had some impairment of hearing and appeared to be retarded mentally. Their voices were hoarse, and there was some stuttering. Their teeth showed decay. The thyroid enlargement was greater on the right in one and on the left in the other. Both showed involvement of the laryngeal nerves on both sides.

Laboratory Examination.—The basal metabolic rates were —2 to —8. Roentgenograms showed the trachea of each compressed from both sides. In one the trachea was displaced to the left. The bones of the hands showed retarded fusion at the epiphyseal lines, with bones shorter than normal. The results of the remainder of the laboratory examination were within normal limits.

Treatment.—Both patients had subtotal thyroidectomies, from which they made good recoveries.

Postoperative Progress.—The wounds healed without complication. Examination of the vocal cords showed some improvement. The basal metabolic rate was —12 in one and —24 in the other.

Fig. 3.—Photomicrographs of the twins shown in Figure 1, showing similar papillary adenocarcinomas. Sections taken from right lobes of thyroids.



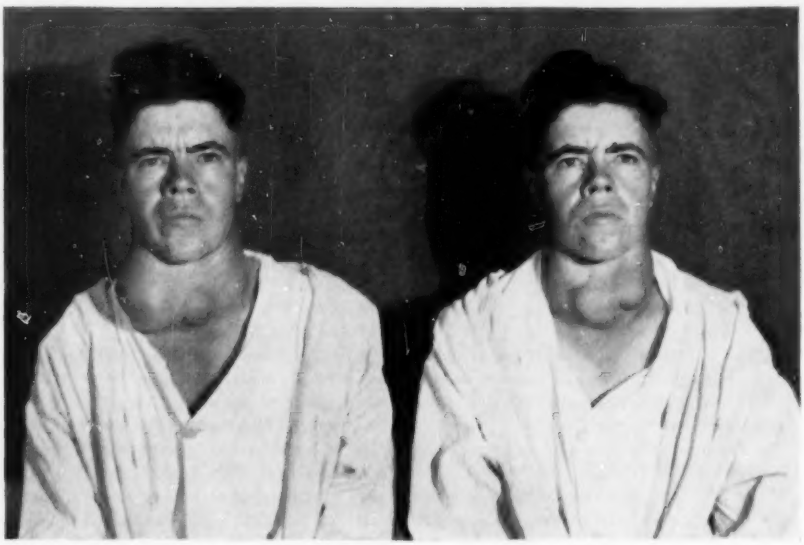
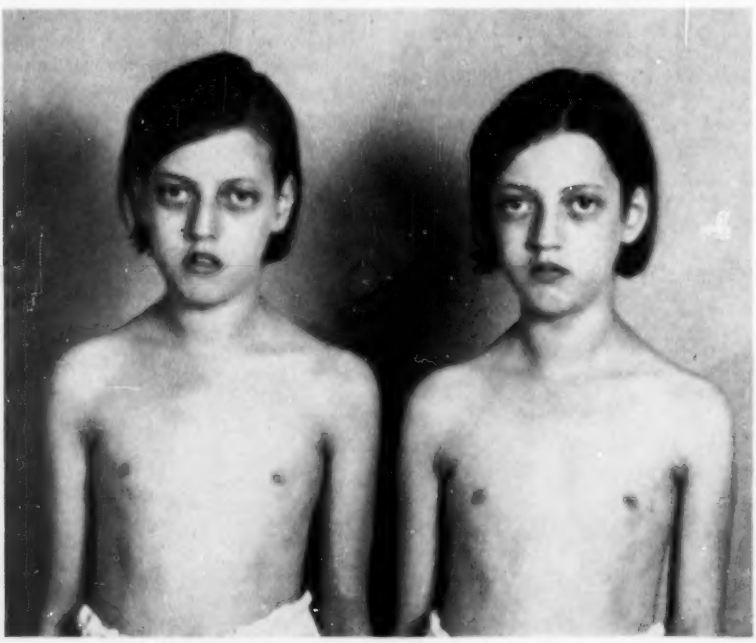


Fig. 4.—Identical male twins 30 years of age, showing nontoxic nodular goiters and evidence of cretinism.

Fig. 5.—Identical female twins, 10 years of age, showing exophthalmic type of goiters.



These twins could not be traced, and their progress has not been known since they left the hospital.

Diagnoses.—The diagnoses were nontoxic nodular goiter, with some cystic degeneration, and cretinism.

TWINS 3.—These identical twins, R. and F., were sisters, aged 10 years. They were admitted to the University of Kansas Hospitals in 1930. Enlargement of the neck and prominent eyes were noted in R. at 8 years of age and in F. at 10 years of age. Typical exophthalmic goiters were diagnosed in both (Fig. 5). They were treated conservatively for two years. At age 12, subtotal thyroidectomies were done at the Mercy Hospital, in Kansas City, by Dr. L. P. Engel. Examination six years later showed good results from the operations and normal development. (These twins were reported in detail by Dr. F. C. Neff in 1932.[8])

COMMENT

Disease of the thyroid in twins is rare. Sporadic cases have been recorded throughout the world, but the total number is not great. Siemans[9] has collected the case reports of thyroid disease in twins up to 1924. In 41 pairs of identical twins only 1 pair showed different thyroid development. Sixteen had fairly large strumas. Twenty-two had nonvisible but palpable strumas, and two with symptoms of thyroid disease had both nonpalpable and nonvisible thyroids. There were 29 pairs of unidentical twins. Four of these showed fairly large visible strumas; nine pairs each had one visible and the other nonvisible palpable strumas. Nine pairs had nonvisible but palpable strumas, and seven pairs had nonvisible and nonpalpable strumas. It is concluded from this study that there is a wide range of development of thyroid disease in both identical (monozygotic) and unidentical (dizygotic) twins. It is apparent also that in identical twins the development of similar thyroid in each twin is much commoner than in unidentical twins. A summary of the entire group of 70 pairs of twins shows that of the 41 identical twins, 1 pair showed different thyroid development, whereas, in the 29 unidentical twins, 13 showed identical strumas and 16 had different development of the thyroid. The difference in identical and unidentical twins is graphically shown in Table 2.

Carmena[10] recorded two pairs of identical female twins, aged 18 and 37, with hyperthyroidism. He collected from the literature in 1949 twelve other pairs of identical twins with goiters. There are, no doubt, other cases of twins with goiters reported which we have not found.

We have not found reported any pair of twins with thyroid carcinoma. Sprague † has given us a personal report of a pair of identical female twins, aged 22, one of which was operated upon for papillary carcinoma of the thyroid. The other twin had no evidence of thyroid disease at the time of operation upon her sister, April 8, 1953.

TABLE 2.—*Summary of Thyroid Disease in Seventy Pairs of Twins*

	Pairs
Identical Twins	
Fairly large strumas	16
Different thyroid development	1
Nonvisible but palpable strumas	22
Nonvisible and nonpalpable thyroids	2
Unidentical Twins	
Fairly large strumas	4
Visible struma in one with nonvisible but palpable struma in the other	9
Nonvisible but palpable strumas in both	9
Nonvisible but palpable struma in one; nonvisible and nonpalpable struma in the other	7

SUMMARY AND CONCLUSIONS

Three pairs of identical twins have been studied at the University of Kansas Medical Center. They had three types of thyroid disease. These types were carcinoma, nontoxic nodular goiter with cretinism, and exophthalmic goiter.

These twins were all operated upon for their thyroid diseases. There have been no deaths. Twenty months have elapsed since the operations for carcinoma, with no evidence of recurrence. The twins with nontoxic nodular goiters and cretinism could not be traced. Six years after operation the twins with exophthalmic goiter were in good health and developing normally.

We have not found a single report of carcinoma of the thyroid in identical twins in

† Sprague, F. H.: Personal communication to the authors.

medical literature. It is possible that such reports may be buried in reports of carcinoma of the thyroid in general and have not been found.

If disease of the thyroid develops in one of identical twins, it may be expected to develop in the other at the same or some future time.

If one identical twin develops carcinoma of the thyroid, we believe that there is justification for exploration of the thyroid of the other twin even if clinical evidence of disease is not present. If evidence of nodular goiter is found, subtotal thyroidectomy is indicated even if carcinoma has not developed.

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Cancer of the Thyroid

J. E. KEARNS, M.D.
HARWELL G. DAVIS Jr., M.D.
and
RUTH B. BALKIN, M.D., Evanston, Ill.

Three years ago we reviewed our experience with nodular thyroid glands.[1] We concluded, then, that the usefulness of any histologic classification of cancer of the thyroid is inversely proportional to its complexity. Histologic examination is no more valuable a source of information about tumors of the thyroid than a complete gross examination. It was demonstrated that all histologic types could be found in the majority of cancers as well as in multiple metastases. From this experience we have developed the following simple classification of cancer of the thyroid gland used in this discussion:

1. Adenocarcinoma
   (a) Directly invasive
   (b) Lymphatic invasive
   (c) Hemangioinvasive
2. Undifferentiated carcinoma
3. Sarcoma

INCIDENCE

In an eight-year period we have done 300 thyroidectomies upon clinically abnormal glands. Thirty-one were cancers, an increase of 10%. The distribution of these is shown in Table 1. We are including for discussion another 20 cancers which have been treated by us but upon which the primary surgery was performed elsewhere. The age and sex incidence of these is similar to the first group, making a total of 51 patients with cancer of the thyroid. All had a previous history of goiter, none of hyperthyroidism.

TABLE 1.—*Age and Sex Incidence of Cancers of Thyroid*

	Patients with Clinically Abnormal Glands		
	Total	With Cancer	Per Cent with Cancer
Under 12 yr.	4	4	100
Males	45	8	18
Females	251	19	7.5
Total	300	31	10

DIAGNOSIS

Diagnosis is made only by biopsy, which must be either lobectomy or subtotal thyroidectomy. Gross findings at operation correlated with pathologic examination serve best to make the diagnosis of cancer. This is particularly true when the differentiation must be made between carcinoma and "thyroiditis." Frozen sections done on inadequate bits of tissue usually have been more confusing than helpful (Fig. 1).

TREATMENT

If the lesion has been removed completely for biopsy and there is no evidence of lymph gland involvement, there is no further treatment. If metastases are present at the primary operation, we do a total thyroidectomy and radical neck dissection with removal of homolateral nodes.

If gross blood vessel involvement is present, extensive blood vessel resection is done to secure the jugular and inferior thyroid veins as low as possible.

The undifferentiated tumors usually involve the entire thyroid gland. In these, we remove as much of the gland and adjacent tissue as is feasible.

When large masses of tumor require an intrathoracic approach, we have used sternal,

From the Departments of Surgery and Pathology, Evanston Hospital Association and Northwestern University Medical School.

Read at the Sixty-Second Annual Meeting of the Western Surgical Association, Colorado Springs, Colo., Dec. 4, 1954.

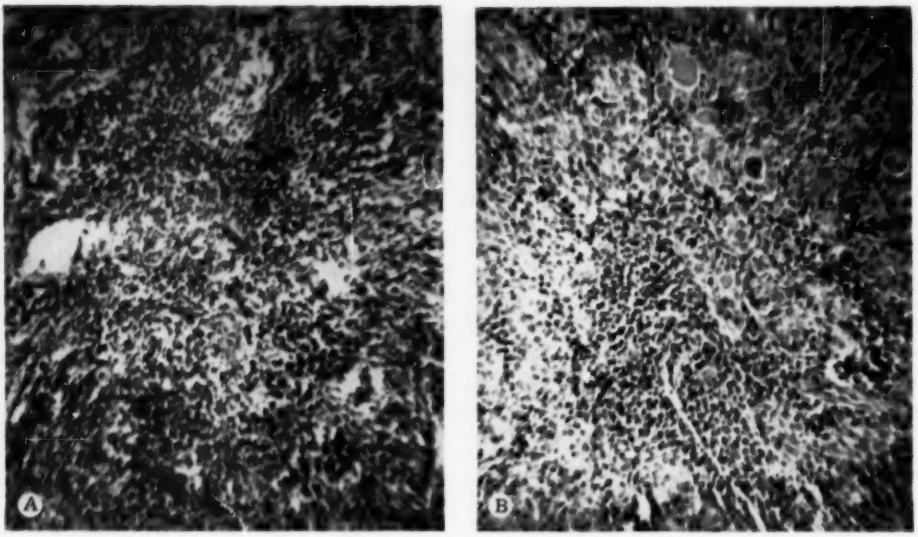


Fig. 1.—*A*, frozen section misdiagnosed as carcinoma; *B*, paraffin section: "thyroiditis."

clavicular, and rib resections in addition to the radical neck dissection. The choice of thoracic procedure depends on location of the masses.

Radioautographs are made routinely on all thyroid tissue removed. The primary tumor may not take up I^{131}, but the metastases may. Therefore, one can not predict the actual therapeutic effectiveness of this drug from its absorption by the primary tumor. Metastases in bone have demonstrated better uptake than those in the lungs.

Instead of massive dosage of I^{131}, continued small doses (40 to 60 mc.) are given over a long period of time. Treatment has been interrupted because of effects on other tissues, but there has been no instance in which the therapy was more malignant than the disease. Two patients who have received more than 900 mc. have since had normal children. The children treated have been given desiccated thyroid and have progressed normally toward maturity. This type of therapy is controlled by external survey and urinary excretion studies. When lesions fail to absorb radioactive iodine, we use the following methods to stimulate uptake: thyroid-stimulating hormone, thiouracil, and desiccated thyroid. The latter has been the most effective. X-ray therapy is our choice in the undifferentiated tumors.

TABLE 2.—*Summary of Data on Patients with Cancer of Thyroid*

Age Range, Yr.	No. of Patients				
	Total	Living and Well	Living with Cancer	Had Many Operations	Dead
0-15	4	4	.....	.....	..
16-30	22	21	1 (4%)	4 (18%)	..
31-55	14	9	3 (21%)	10 (70%)	2*
56-75	11	1	5 (45%)†	5 (45%)	5

* Twenty-year survival.
† Thirty-six-year survival.

All 51 of our patients have been operated upon. Approximately 80% have received radioactive iodine; 12%, x-ray therapy, and 8% have received no therapy other than surgery.

RESULTS

Seven patients are dead, all over 50 years of age. Forty-four are alive with or without tumor. Of the seven, one died of massive metastases 20 years after primary operation; one, with an undifferentiated carcinoma died from gastrointestinal hemorrhage. Autopsy demonstrated invasion of neck structures but no distant metastases. Two died from slough-

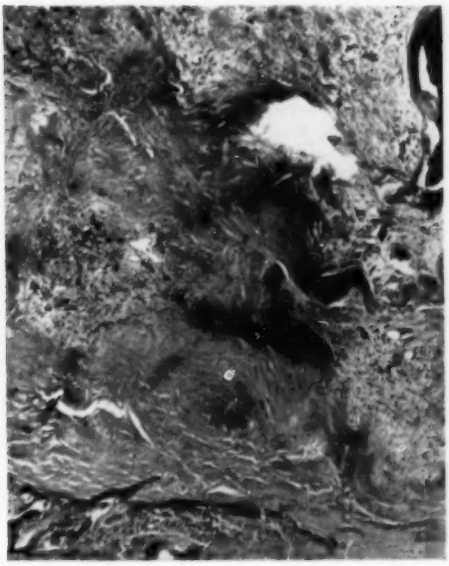
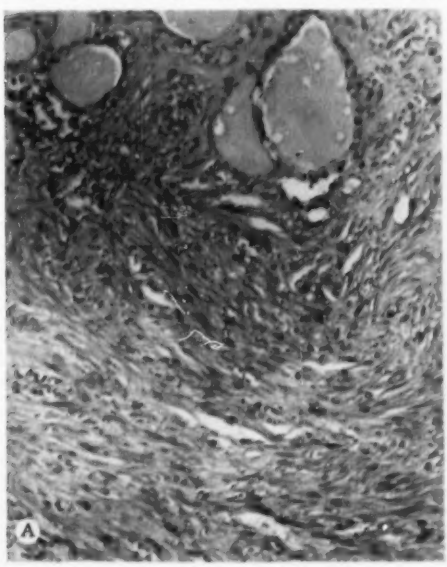


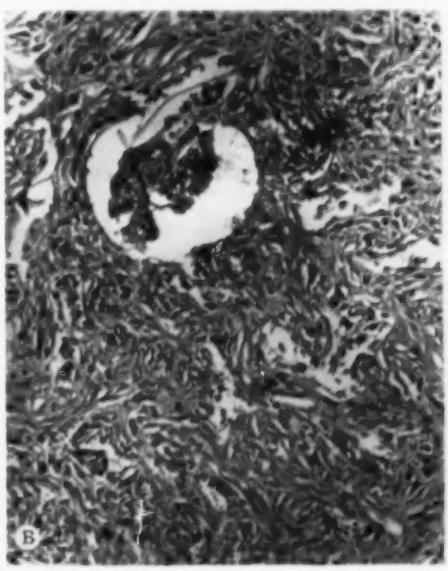
Fig. 2.—Metastasis to bone showing healing after radioactive iodine. Reduced to 1/2 of mag. × 100.

Fig. 3.—Erosion of common carotid artery following operation (reduced to 1/2 of mag. × 35).

ing of a major blood vessel, one after operation, the other after x-ray therapy (Fig. 2). The fifth death was associated with extensive ulceration of the larynx and esophagus following x-ray therapy. The sixth and seventh had sarcoma allegedly primary in the thyroid. At necropsy, one had Hodgkin's disease. The sarcoma was apparently primary in the kidney area (Fig. 3).

Fig. 4.—*A*, fibrosarcoma metastatic in thyroid; reduced to 1/2 of mag. × 250. *B*, fibrosarcoma primary focus in kidney; reduced to 1/2 of mag. × 250.





Of the living patients, four under 15 years of age have survived two to eight years without clinical evidence of carcinoma. Twenty-two patients 16 to 30 years of age are well from 3 to 15 years without cancer. Four have had multiple operations for cancer. Of patients from 31 to 55 years of age there are 12 living, 3 with cancer. Eight have had multiple operations. In the age group 56 to 65 there are six patients; one is living and well 10 years since her operation. The others have survived from 18 to 36 years and are living with cancer. All have had multiple operations, some as many as eight (Fig. 4).

SUMMARY

Review of these cases of carcinoma has demonstrated that distant metastases occurred only after demonstrable blood vessel invasion. Only local metastases resulted from lymphatic invasion, and deaths of patients in this group followed therapy. Hence, we find our grouping useful.

The importance of necropsy examination is obvious from the recorded deaths. Of the seven, in only one was death actually an immediate result of cancer of the thyroid. Three deaths can be ascribed directly to therapy; one, to unrelated disease. Two diagnoses of primary tumor were found to be erroneous. We have found no contraindication to long-continued radioactive iodine therapy if it be carefully controlled. It seems to be most effective in controlling metastases to bone.

Carcinoma of the thyroid usually begins early in life. Survival is long, being dependent upon the rapidity with which blood vessel invasion takes place.

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DISCUSSION

On Papers by Drs. Robinson and Orr and Kearns, Davis, and Balkin

Dr. Willard Bartlett, St. Louis: I share the view so well documented by Dr. Pemberton about 15 years ago that cancers of the thyroid, as a group, offer the most hopeful outlook of all cancers, the great majority being of rather low malignancy and in an accessible location. We all feel almost helpless, however, in contemplating the fate of that small group of patients with highly anaplastic, undifferentiated cancers, of whom nearly all will be dead in less than a year from the appearance of the growth, no matter how treated. I wish to cite a personal experience, not to challenge the validity of such a pessimistic view, but to urge others not to contemplate palliative methods alone upon first consideration of a lesion of this type.

In September, 1950, I first saw a patient, Mrs. G. H., aged 48, who had had exploration of the thyroid and partial left thyroid lobectomy performed elsewhere nearly four months previously, the lesion being grossly confined to the left lobe and being recognized as carcinoma. Sections had shown it to be highly anaplastic, and the patient's husband had been informed that nothing further could be done. The patient came to me complaining of dysphagia, dyspnea, and pain. There was a fixed bilateral mass in the thyroid area and much redness, tenderness, and induration of the skin. There was fixation of the left vocal cord. I hospitalized the patient at St. Mary's Hospital, expecting to have to do an early tracheostomy for the relief of dyspnea and as an adjunct to palliation with x-ray therapy. A review of her previous sections in another hospital confirmed the reported findings of round-cell, utterly undifferentiated carcinoma.

On Sept. 16, 1950, the neck was explored through a collar incision, my intention being to excise only enough of the thyroid mass to get access to the trachea. Gross infiltration of the sternohyoid and sternothyroid muscles was obvious, as was extension into the right lobe from the primary growth in the left lobe, now the size of a lemon. To my surprise, the fixation of the mass deeply was due to the left lobe being wedged under the clavicle, and complete attachment to the carotid sheath had not occurred. Since I could get behind and below the left lobe, therefore, I undertook a radical operation largely in the belief that it would offer far better palliation than the contemplated procedure. The superficial flaps were revised to leave all fat attached en bloc with the cervical fascia, and the skin incision was extended from its left lateral angle upward along the sternomastoid muscle. The left sternomastoid muscle, the left jugular vein and anteromedial aspects of the carotid sheath were included in the block along with the right and left prethyroid muscles and both lobes of the thyroid, plus the subcutaneous fat and the superficial layer of the deep cervical fascia. It is significant that fixation to the trachea anteriorly was due to inflammatory reaction, not to infiltration of the trachea. The most serious fixation was encountered in the hypopharynx on the left, where the growth

was "shaved off" with some trepidation as the last step. The left inferior laryngeal nerve was included in the neoplastic mass. A tracheostomy was performed in anticipation of x-ray therapy, which was started in about two weeks and carried out by Dr. Leroy Sante thereafter.

General and laryngoscopic examinations were made regularly, and no evidence of extension, recurrence, or metastasis had appeared at the time of her death, on May 22, 1952. Complete autopsy failed to reveal evidence of cancer 20 months after the date of radical operation.

I have thought this case to be worth reporting as a reminder that careful exploration may occasionally warrant an aggressive attitude with the salvage of such a patient, even when a false start has previously been made. It is ironical that the surgeon who had pronounced her situation to be hopeless died of an acute coronary occlusion a few months after his abortive exploration, the patient outliving him by more than a year.

DR. R. MORTON BOLMAN, Fort Wayne, Ind.: I cannot resist saying just a word about carcinoma of the thyroid.

I felt all along that my attitude toward thyroid nodules was as aggressive as anybody else's, and last June, while visiting in New York and watching my wife talk on the telephone, I noticed a small mass ascending and descending in her neck while she swallowed, about the size of one's thumb, she being completely unaware of the presence of the mass. That was on a Friday.

On Tuesday, after we returned home, Dr. Lloyd did a total thyroidectomy on her, and on Wednesday the report was papillary cystadenocarcinoma. This was verified by Shields Warren and William Meisner, and three days later she had a radical neck dissection, with no evidence of extension anywhere.

The reason I mention this, with her permission, is because we, along with everyone else, have the experience of hearing our referring doctors say, "The nodule hasn't grown any, it doesn't bother the patient, it isn't toxic," and I wonder if we realize how limited the knowledge of the general practitioner is of the potentialities of thyroid nodules.

Dr. Lloyd and I immediately undertook a vigorous educational program not only among our referring doctors but in scrub rooms and dressing rooms for anybody who would listen, with the result that since June of this year we have been inundated with 63 thyroid nodules, of which 6 were papillary cystadenocarcinomas, including my wife's. Five of those patients had complete radical neck dissection. One of them, who is still in the hospital, is going to have to ride with a total thyroidectomy and x-ray therapy because of the fact that she also had an endometrioma of the rectosigmoid with practically complete obstruction, necessitating a second procedure at the end of 10 or 12 days for relief of her obstruction.

This seems to be a type of carcinoma for which we really have something to offer people. I mention this only because I believe the over-all statistics of cure can be greatly improved upon by people of the standing of this group educating the general practitioners in their vicinity.

I forgot yesterday to thank you for the privilege of being here. May I do that now. Thank you very much.

MR. TOBEY LEVITT, Chicago: I have enjoyed these two papers very much. Dr. Robinson's paper on these three different factors which are genetically important make us realize we must follow the histories of these patients and their relatives.

I would like to ask Dr. Robinson whether he studied, first, geographical factors, diet, goitrogenic factors, and whether they followed the genetic histories of these patients.

I would like to ask Dr. Kearns how he assessed his papillary and his follicular carcinomata, and on what basis the final histological pattern was drawn up.

[Slide] This is a study of 256 thyroid glands with malignant disease. You will note that 20% were papillary tumors. I would like to ask Dr. Kearns why there are so few papillary tumors in England and why there are so many papillary tumors in the United States.

[Slide] In 1948 when I studied these 256 malignant changes of the thyroid, I noted that there were many malignant nodes behind the sternum which were the cause of death, and therefore evolved an operation which I would like to present to you for consideration. Owing to lack of time I will run through the chief features pictorially.

The first stage of the operation, a cerviconodal operation.

The second stage is a thyrocerviconodal excision, and the third stage, a thyrocervicomediastinonodal excision.

[Slide] This shows the concept. There are three steps. (1) Cerviconodal excision: internal jugular lymph node excision in continuity with the regional lymphatics of the thyroid gland. (2) Thyrocervicoconodal excision: total thyroidectomy with removal in continuity of the supraisthmial and infraisthmial lymph nodes. (3) Mediastinonodal excision: a thymectomy, including a removal of the mediastinal lymph nodes in continuity with the thyrocervicoconodal excision. The sternal split operation required is essentially an easy operation. The consecutive steps of the new operation evolved are here explained pictorially by means of 20 lantern slides.

DR. ERICH A. QUER, Kansas City, Mo.: Mr. President, Members and Guests.

First I wish to thank the Association for the privilege of attending this very fine meeting and allowing me to comment on this excellent paper.

I would like to give you briefly our experience in Kansas City with carcinoma of the thyroid and reemphasize the point that Dr. Bolman made about educating our so-called "local M.D.'s" that thyroid nodules are dangerous. The following two tragic cases, which we saw within a month just recently, should help to further place emphasis upon this problem.

The first one, a 21-year-old girl, who was a secretary to two rather young general practitioners near our city, noticed a nodule in her thyroid and confronted both of these doctors with it. They adopted a "watch and see" attitude, and so two years later she presented herself at our office, at which time she not only had the thyroid nodule but also a palpable lymph node [illegible] below it. Dr. Claude Hunt, of this association, operated upon this woman and found a carcinoma of the left lobe of the thyroid with extensive lymph node involvement, even into the superior mediastinum. Consequently a radical operation was done which included a superior mediastinal dissection, which we feel is very important in these cases, because of the anatomical arrangement of the lymph nodes and their drainage pathways.

The second case is an 11-year-old boy who had several "lumps" in his neck. His M.D. did not attach any significance to these and watched them for about one year. The boy was finally referred to us by another doctor and was found to have multiple discrete nodules throughout all the triangles of his neck. One of these was studied by biopsy and proved to be a papillary carcinoma of the thyroid gland. On his 12th birthday we performed a one-stage total thyroidectomy and bilateral neck dissection with preservation of the sternocleidomastoid muscles and the internal jugular veins. A concomitant tracheostomy was performed because of the extensiveness of the dissection and because we wanted to give this boy heavy postoperative irradiation. In spite of this the boy is in the hospital again now (February, 1955) because we had to remove several palpable nodes from his right lateral and posterior triangles of his neck, some of which contained carcinoma.

In reviewing our last 100 consecutive cases of so-called discrete thyroid nodules we found 11 cases which proved to be carcinomatous on microscopic examination.

I know that the members of this association realize the importance of thyroid nodules, but it is also up to the members of this Association and societies like this to educate our friends and referring physicians who see these problems early and impress upon them the importance of getting these thyroid nodules out.

Thank you again for the privilege of being here.

DR. DAVID W. ROBINSON, Kansas City, Kan.: I fear I can answer very little of the question Mr. Levitt poses, except to say that to my knowledge each set of these three sets of twins had lived under the same conditions of environment and diet all their lives, and we know of nothing specific in their genetic pattern.

The only difference was in the set of twins whom I showed first, the 24-year-old women. One had gone into nursing and the other had studied to become a teacher, which I think is due to a rather definite different environmental factor having no bearing on the situation.

It should be pointed out that identical twins, for all practical purposes, are one and the same person. They are simply a fertilized egg that was split and that developed two people from the same beginning.

Thank you.

DR. J. E. KEARNS, Evanston, Ill.: To answer Dr. Bartlett's question, we have had four undifferentiated cancers of the thyroid, and all have had extensive surgery, as extensive as we could do on the neck. All have had tracheotomies postoperatively during their x-ray therapy. Two are living and probably have cancer, one for two years and the other for about one year. Of the two who died, one was the patient who died from a gastric hemorrhage and who had only local invasion of the neck from her cancer. The other was one, seen at necropsy, who died from a sloughing following x-ray therapy. This, we felt, was due to the tracheotomy tube. We now use plastic tracheotomy tubes in all these patients at the time of therapy.

In answer to Mr. Levitt, we do not recommend a histological classification. That very easily answers his question.

SURGERY ILLUSTRATED

Peptic Ulcer in Children

F. K. ALEXANDER, M.D., Darby, Pa.

Peptic ulceration in children is infrequent statistically though recent publications in the specialty journals have given emphasis to the occurrence and diagnosis of peptic ulcer in infants and children through the age of 16 years.[1] Recent reviews of the literature of gastric ulcer in children reveal less than 50 reported cases, with the majority of the reports dealing with 1 and 2 cases. This would indicate the rarity of the lesion but raises the question as to whether the incidence based on reported cases represents a true incidence of the disease. This is given further emphasis by autopsy statistics on children dying from all disease in which an incidence of peptic ulceration is given between 0.1 and 0.2%. It is well recognized that autopsy statistics based on hospitalized patients never represents a true incidence of any disease in the general population and would suggest a far greater incidence of unrecognized and undiagnosed cases.

Using autopsy statistics as a basis for computation, Goldsberry [2] suggests there are approximately 50,000 undiagnosed gastric ulcers between the age of one and six years. If one includes all age groups from infancy to 16, the potential assumes proportions of such magnitude as to merit continuing attention from the clinician and radiologist.

The incidence of duodenal ulcer in children is likewise difficult to appraise statistically, although the literature contains frequent reports of series of cases diagnosed at autopsy by surgery or radiologically.

From Department of Radiology, Fitzgerald Mercy Hospital, Darby, Pa.

Read before the Section on Radiology at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 25, 1954.

The autopsy statistics suggest an incidence varying from 1 to 1.5%. Again the inherent error of autopsy figures for determining the frequency of a disease must be remembered to obtain a more realistic appraisal of the clinical incidence.

The paragraphs above would emphasize the realization that peptic ulcer, like many other medical entities, is uncommon but does exist and if a high index of suspicion is exercised clinically the likelihood of more frequent diagnosis would result.

The ratio of duodenal to gastric ulcer from existing reports in the literature would be in the ratio of more than 10 to 1. Duodenal ulcer is more prevalent in the male, while gastric ulcer is approximately evenly divided between the sexes.

While it is probably impossible to estimate the incidence of peptic ulceration at various age groups, one seems to notice a greater frequency of reports of the disease in patients during the years when the child is under the care of a pediatrician, when recurring gastrointestinal symptoms would more likely be investigated.[3] These reports also contain a

Fig. 1.—Twenty-day-old infant brought into the hospital with symptoms of regurgitation of most feedings for the past few days. Vomitus at first contained greenish mucus; later, bright red blood. A profuse intestinal hemorrhage occurred, and the infant died. Autopsy revealed a perforated duodenal ulcer on the posterior wall. Illustration shows section taken through the perforated ulcer (reproduced from Alexander,[10] *Radiology*, published by The Radiological Society of North America, Inc., Syracuse 2, N. Y.).

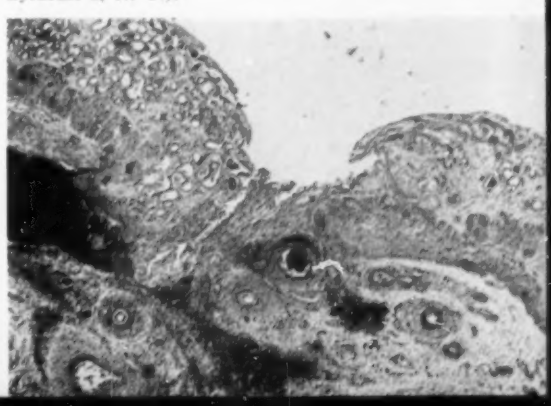


Fig. 2*A*.—A 2-month-old infant girl with history of feeding problem difficulty and failure to gain weight. Infant began to vomit feedings, and later vomitus contained bright red blood. The patient showed evidence of considerable blood loss and was given several blood transfusions before x-ray studies. These showed persistent bulbar deformity with niche defect at base of bulb.

Fig. 2*B*.—(Same case as Fig. 2*A*.) After repeated blood transfusions and medical dietary treatment, patient continued to improve. Patient was again x-rayed, four months after the original study, and at this time the duodenal bulb showed no defect or evidence of inflammatory changes.

Fig. 3*A*.—Five-year-old boy with long history of abdominal pain, nausea, and vomiting of thick yellow mucus. There was distress following the eating all types of food. X-ray studies revealed a marked bulbar spasm and deformity with a persistent niche defect of ulcer. Reproduced from *Radiology*, published by The Radiological Society of North America, Inc., Syracuse 2, N. Y.

Fig. 3*B* (same case as Fig. 3*A*).—Six months later. All symptoms were somewhat improved but particularly the abdominal pain. Episodes of nausea still occurred, and dietary breaks usually resulted in vomiting. X-ray studies reveal less bulbar deformity though still irritability with the niche defect well shown. Reproduced from Alexander [10] *Radiology*, published by The Radiological Society of North American, Inc., Syracuse 2, N. Y.

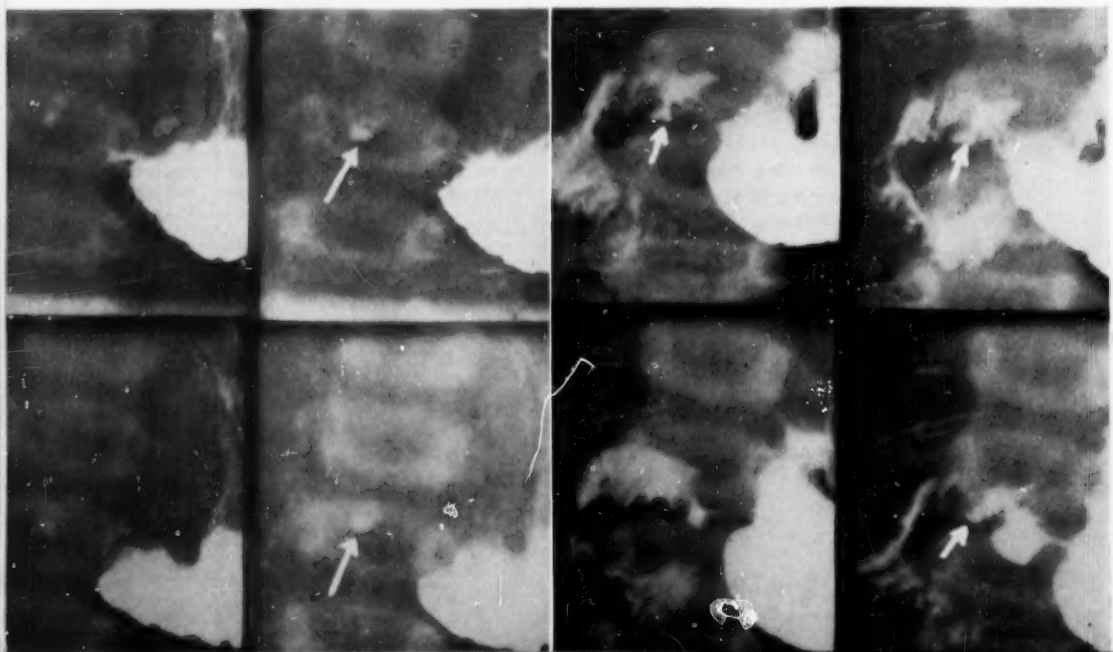


Fig. 4*A*.—Twelve-year-old boy with long history of generalized abdominal pain, nausea and vomiting which were intermittent in character. X-ray study, elsewhere, showed a marked antral mucosal irregularity and irritability plus duodenal irritability and deformity and a typical niche defect of ulcer. This patient was a heavy user of alcohol and tobacco and limited his food and excesses only while having acute gastric distress. He would not cooperate medically.

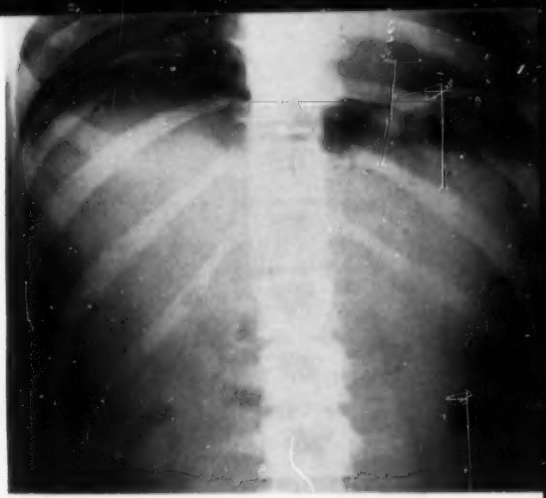


Fig. 4*B* (same case as Fig. 4*A*).—About four years later, patient now 16 years old, admitted to another hospital after a severe attack of abdominal pain, nausea, and vomiting. No studies were done, and the patient was then admitted to our hospital, where an abdominal series showed evidence of a perforated viscus and peritonitis.

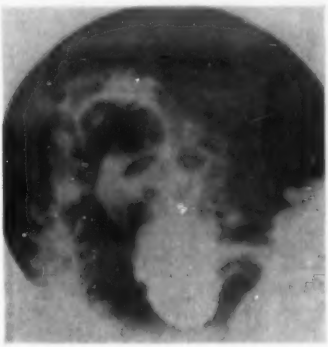
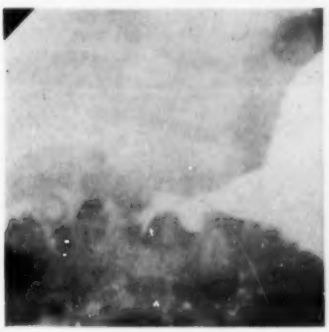


Fig. 4*C* (same case as Fig. 4*A* and *B*).—X-ray studies two weeks after admission showed evidence of a marked antral and bulbar irregularity with an appearance suggesting the site of the perforation on the greater curvature of the bulb.

Fig. 5*A* and *B*.—Seventeen-month-old baby brought into the hospital by mother because of currant-jelly-colored clots and bright red blood coming from the mouth and tarry stools. Patient had recently fussed about eating because of apparent distress and had failed to gain weight. There had been no previous symptoms suggesting gastrointestinal tract disease. Hemoglobin 42%. Patient transfused several times and showed definite improvement. No further bleeding. Patient began to eat better. X-ray studies showed a gastric ulcer on the greater curvature of the stomach. Conservative medical regimen instituted.

Fig. 5*C* (same case as Fig. 5*A* and *B*).—Patient continued to improve while in the hospital and was apparently a well child. X-ray studies, 17 days later, showed no evidence of the gastric ulcer. Follow-up inquiries reveal child to be symptom-free.



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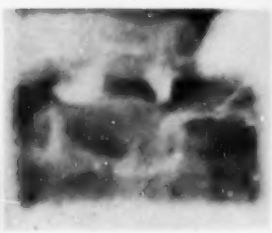

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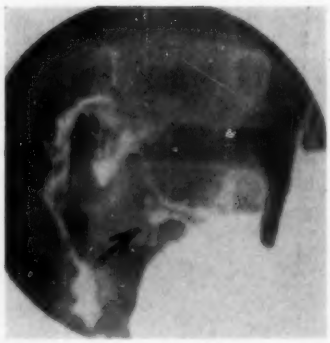
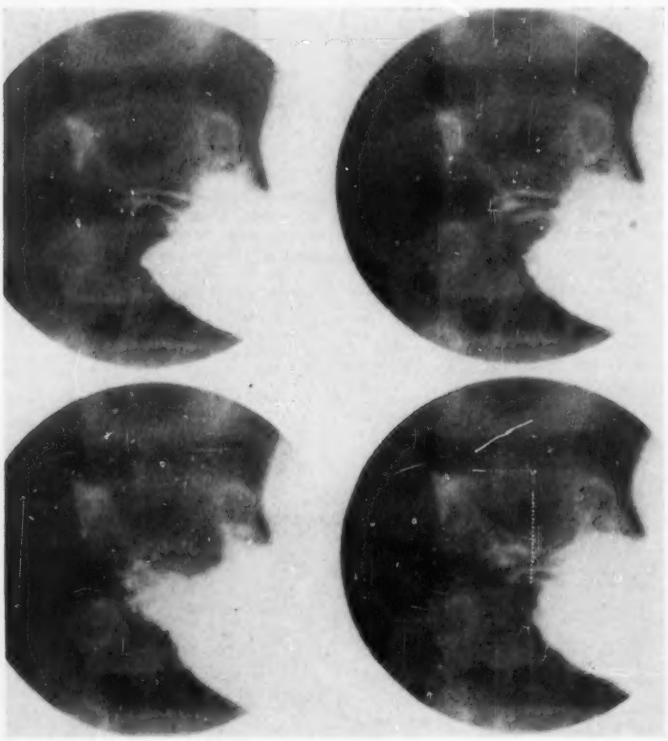


B

Fig. 6*A* and *B*.—A 7½-year-old boy with a history of severe cramp-like bouts of high abdominal pain, nausea, and vomiting for several weeks. No qualitative food history. Laboratory studies were essentially negative. The clinical diagnosis was "peptic" ulcer. X-ray studies showed a dilated stomach with a nonopaque residue, marked antral spasm, and persistent niche defect of ulcer.

Fig. 6*C* (same case as Fig. 6*A* and *B*).—Patient was placed on a conservative medical regimen which resulted in some improvement, although dietary breaks resulted in reappearance of pain and nausea and vomiting. X-ray studies, six weeks after the first examination, still revealed marked antral spasm and deformity, though the niche defect is not as prominent as before.

C



high proportion of the acute or emergency symptom complex of gastrointestinal tract disease, with symptoms of hemorrhage or perforation, or both. The dearth of reports on peptic ulceration in patients beyond the age of 16 years to early adult life would suggest the likelihood that poor medical supervision is responsible in this age group rather than absence of the disease.

ETIOLOGY

The literature on the etiology of peptic ulceration in adults is voluminous.[4] Some of the reports are based on careful observations in rigidly controlled experiments in which numerous agents have produced peptic ulceration while other reports are speculative and inconclusive. It is reasonable to admit the potential of many factors which can cause peptic ulceration in man and in experimental animals with as yet no common denominator and conclude that the cause of peptic ulcer is as yet an unsolved problem in adults as well as in children.[5]

SYMPTOMS

The symptomatology of peptic ulcer in children would seem to be somewhat dependent upon the age of the patient rather than the location of the lesion. Abdominal pain, nausea, anorexia, and vomiting are frequently found in the older child, while the feeding problem complex group of symptoms are found in the infant and young child with bleeding and perforation (Bird) statistically more likely at this age.[6]

Peptic ulcers in infancy may be associated with extensive hemorrhage, acute perforation, and shock, and the events may occur with great rapidity, resulting in death before a diagnosis is made or treatment begun. These cases may have symptoms relating to feeding problems, with irritability, vomiting, refusal to eat, and failure to gain weight. Occasionally, no recognized evidence of gastrointestinal disease is present before a massive hemorrhage or perforation occurs.

Blood in the vomitus is not uncommon and occult blood may be found upon stool examination. The distribution of the pain in the older child is not significant or diagnostic. It may be generalized throughout the abdomen although occasionally periumbilical or epigastric in origin.[7] There does not seem to be any noticeable difference in area of pain distribution or time of occurrence to distinguish symptomatically between gastric and duodenal lesions. The adult ulcer patient's complex of "pain, food, ease" has not been a prominent pattern, though occasional patients derive some relief from warm milk feedings. Chronic appendicitis has been held responsible and children have been subjected to unnecessary surgery without benefit. Abdomi-

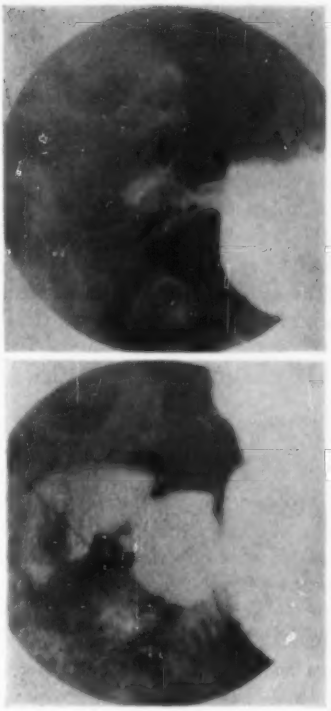


Fig. 7*A* and *B*.—Six-year-old girl with long history of "feeding problem" difficulties since infancy. Thought to be an allergic child and placed on goat's milk until after 14 months of age. All solid foods caused distress, with epigastric pain and vomiting. Child undernourished and underdeveloped because eating caused distress. Had a very severe attack five days previous to x-ray studies which lasted 10 hours, with much pain, nausea, and vomiting. All laboratory studies essentially negative. X-ray revealed marked intermittent antral spasm and deformity with niche defect of ulcer. Later, antrum relaxed somewhat and ulcer niche showed more prominently.

nal migraine, food allergy, mesenteric adenitis, and dietary indiscretions resulting in a gastroenteritis have all been considered likely diagnoses and frequently with justification because of the confusing pattern of the symptomatology and the difficulty of the differential diagnosis.[8] That peptic ulcer deserves to be considered in the differential diagnosis would seem to be indicated by the continuing frequency of reports from the pathological, surgical, and radiological literature, and the plea is made for more frequent gastrointestinal x-ray examinations in children who present similar complaints.[9]

X-RAY APPEARANCE

The x-ray diagnosis of peptic ulcer in children is dependent upon the presence and demonstration on films of a niche defect in the wall of the stomach or duodenal bulb. Associated with the presence of niche defect is the occurrence of irritability and spasm of the adjacent stomach or bowel wall. These findings are most constant and prominent. One also sees evidence of pylorospasm, and preliminary screening frequently shows a nonopaque fluid level or large amount of gas in the stomach. This should raise one's index of suspicion when examining a patient presenting symptoms of repeated gastrointestinal tract upsets in order that a small ulcerative lesion is not overlooked. The niche defect may be large or small, and the associated irritability does not seem to be dependent upon the size of the ulcer or its location. The type of bulbar deformity usually associated with duodenal ulcers in adults is not seen in young children, although it may be seen in the older child who gives a history similar to the chronic adult ulcer syndrome. Careful screening and spot-filming of the stomach and pyloroduodenal region presents some difficulties when examining children, owing to the difficulty of gaining the patient's confidence and cooperation.[10] The experienced examiner will develop a suitable approach to the problem and, once solved, the difficulty of the examination will be no greater in infants and children than in adults. It should be reemphasized that good spot films obtained during screening are necessary to diagnosis or to exclude the presence of peptic ulceration.

Not all patients with antral, pyloric, or duodenal spasm or deformity will show a niche defect of ulcer. As so frequently happens in radiology, the absence of the characteristic findings of a disease presents the greatest difficulty in diagnosis. When a niche is seen, the patient has an ulcer. When the associated findings of antral pyloric or duodenal spasm are present in a patient with gastrointestinal tract symptoms which are repetitive and an ulcer cannot be demonstrated, one probably is justified in assuming the presence of an intrinsic inflammatory process on the basis of antral gastritis or duodenitis. Small shallow superficial erosions not deep enough in the mucosa to collect barium may be present but cannot be seen on the screen or films. Patients with this type of findings are frequently encountered and the pathological and symptom complex of antral gastritis and duodenitis have been well described by Rivers.

SUMMARY AND CONCLUSIONS

The incidence of peptic ulceration in infants and children formerly held to be a rare disease statistically is assuming greater significance owing to the increasing number of case reports appearing in the literature.

Bleeding ulcers from the intestinal tract and symptoms of acute perforation are surgical emergencies with well established criteria for procedural processing. The fact that these symptoms can and do occur in infants and children is receiving the greater attention the seriousness of the lesions require.

Recurrent abdominal pain, nausea, vomiting, and anorexia in the older child and the "feeding problem" group of symptoms in the infant and young child should include peptic ulcer in the differential diagnosis with investigation directed to prove or disprove this possibility.

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Arterial Curettes

ERNEST B. EMERSON Jr., M.D., Rochester, N. Y.

In recent years both vascular surgeons and general surgeons have become interested in the surgical management of large arteries obstructed by thrombi. The operation of endarterectomy has met with varying degrees of success, the end-results depending on factors too numerous and too complicated to come within the scope of this paper.

However, experience with endarterectomy has shown that there is a very real need for instruments which will facilitate the technique and cut down on the operative time. With this in mind, the present work was undertaken at the request of Dr. W. J. Merle Scott. The arterial curettes presented are the result of about two years' work by trial and error as there was no previous basis from which to work.

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CONCLUSIONS

A set of five arterial curettes is presented. They have been found satisfactory for the purpose for which they were designed, i. e., endarterectory. With them portions of thrombus up to 7 in. (17 cm.) in length have been removed in one piece.

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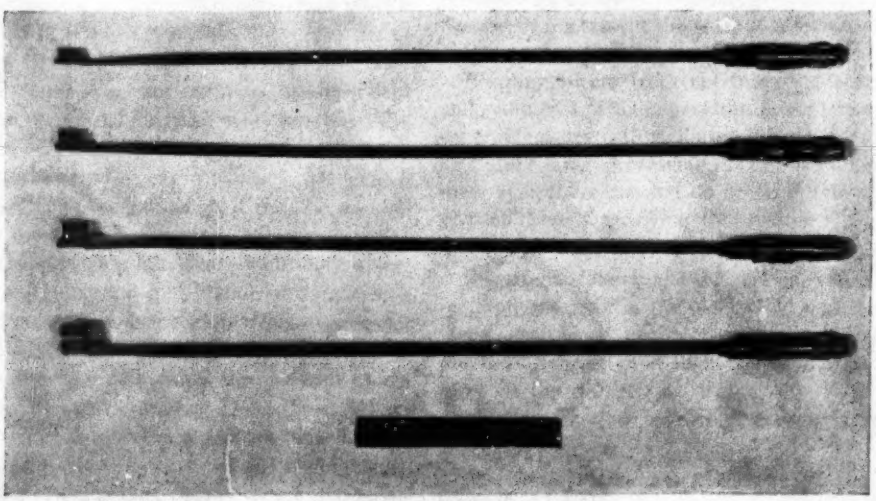

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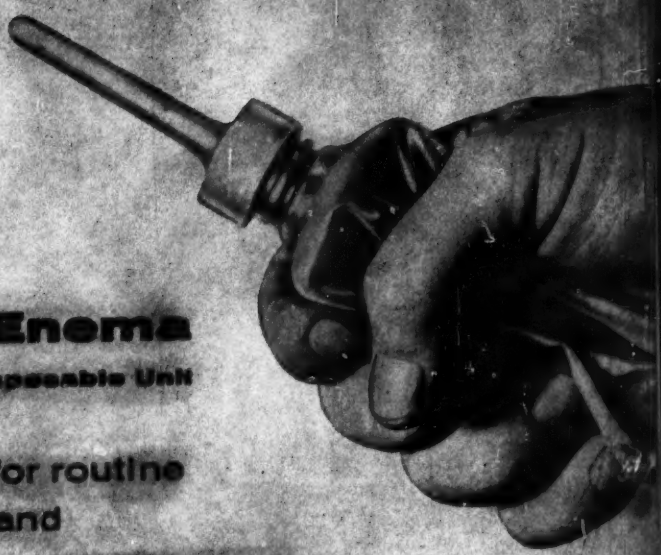

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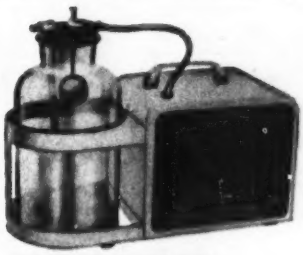

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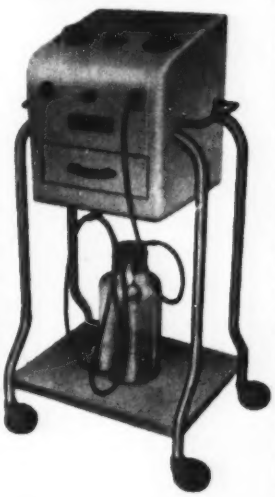

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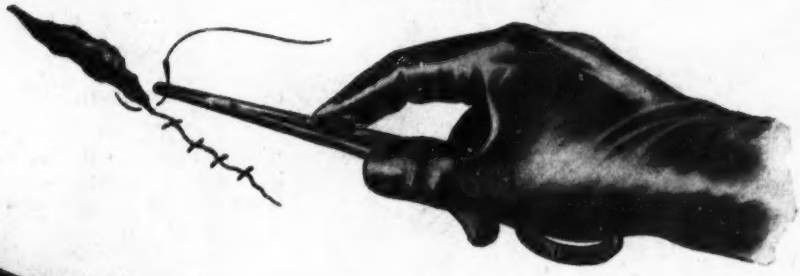

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